

JOURNAL
OF THE
American Veterinary Medical Association
FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit: Mich.

T. E. MUNCE, President, Harrisburg, Pa.

M. JACOB, Treasurer, Knoxville, Tenn.

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Vol. LXXIV, N. S. Vol. 27

February, 1929

No. 3

A PRETTY RACE

In all probability none but those intimately in touch with the situation have been able to appreciate the friendly contest that is being fought by a half-dozen states for supremacy in the matter of membership strength in the A. V. M. A. In 1925 Iowa wrested this honor from Illinois and held first place through 1926, 1927 and 1928, as shown by the annual reports of the Secretary for the years named.

Iowa's jump into first place was unquestionably the result of the meeting in Des Moines, in 1924. The large number of new members admitted at Des Moines was not reflected in the annual report of that year, but one year later. No states made outstanding gains in 1926, but in 1927 Pennsylvania made a very healthy growth, undoubtedly helped by the meeting in Philadelphia that year.

Last year Ohio was the star performer, but without the stimulus of a convention. On the other hand, the Buckeye State had a two-man team that was hard to beat—President Hilty and Resident Secretary Swinehart. Between them they finished the year with sixty new members to the credit of Ohio.

The following figures contrast the relative standing of the six leading states at the present time, as compared with the standing of these same states in 1923. The figures for 1929 give effect to all applications now on file, some of which will be completed

March 1 and the others April 1. We have gone back to 1923, because that year is the earliest for which accurate statistics are conveniently accessible.

<i>State</i>	<i>1923</i>	<i>1929</i>	<i>Change</i>
Iowa.....	269	271	+ 2
California.....	154	266	+112
Pennsylvania.....	178	262	+ 84
Illinois.....	341	259	- 82
Ohio.....	248	258	+ 10
New York.....	224	254	+ 30

The foregoing table shows several interesting points. First of all, there are six states making bids for premier honors, as far as A. V. M. A. membership strength is concerned. Second, it is plainly apparent that California is in the running, having advanced from ninth* place in 1923, to second place at present, just a step behind Iowa. Third, Pennsylvania, with a net growth of 84 members in six years, will have to be given serious consideration.

Someone may ask, "What about our smaller states?" In anticipation of this question, we have prepared a few figures, going to the other end of the table for our data—the six states with the smallest A. V. M. A. membership. Here is how they stood in 1923 and as they stand at present:

<i>State</i>	<i>1923</i>	<i>1929</i>	<i>Change</i>
Arizona.....	7	7	0
Rhode Island.....	7	7	0
Wyoming.....	12	7	- 5
New Hampshire.....	11	8	- 3
Nevada.....	8	9	+ 1
Delaware.....	9	13	+ 4

In these six states we have a net loss of three members over the six-year period. In several of these states, Nevada and Delaware, for example, we have a practically 100 per cent membership, which is more than can be said of any of the larger states.

It is a pretty race. May the best state win.

EXECUTIVE BOARD ELECTIONS

The polls of the primaries in connection with the Executive Board election being held in District No. 2 were closed on January 12, 1929, and the ballots canvassed by a board of tellers, consisting of Dr. C. W. Eddy, Dr. C. M. Hamilton and the Secretary-Editor. There were 255 ballots cast and these con-

*Indiana, Kansas and Missouri, in addition to the five other states named in the table, had more members than California in 1923.

tained the names of 22 nominees. The following five stood highest in the list and were declared nominated:

ALTHOUSE, E. P. Sunbury, Pa.
Practitioner. Graduate of the University of Pennsylvania, 1903.
Joined A. V. M. A., 1903. Secretary of Section on General Practice since 1926.

DE VINE, J. F. Goshen, N. Y.
Consultation and research work. Graduate of American Veterinary College, 1898. Joined A. V. M. A., 1899. Member of Committee on Abortion, 1919-1925.

HOLLINGWORTH, W. G. Utica, N. Y.
Veterinarian, Bureau of Health, Utica, N. Y. Graduate of American Veterinary College, 1884. Joined A. V. M. A., 1885. Member of Committee on Legislation, 1915-1916; resident secretary for New York, 1918-1919.

MARSHALL, C. J. Philadelphia, Pa.
Professor of Veterinary Medicine, University of Pennsylvania. Graduate of University of Pennsylvania, 1894. Joined A. V. M. A., 1895. Vice-president, 1903-1904 and 1908-1909; secretary, 1910-1913; president, 1913-1915.

UDALL, D. H. Ithaca, N. Y.
Professor of Veterinary Medicine and Hygiene, Cornell University. Graduate of New York State Veterinary College at Cornell University, 1901. Joined A. V. M. A., 1913. Member (1923-1927) and chairman (1927-1928) of Committee on Intelligence and Education.

The ballots were prepared and mailed the same day to all paid-up members located in the states comprising District No. 2 (Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Delaware and Pennsylvania). It is interesting to observe that the outcome of this primary election was almost the same as in the election held in the same district, about eight months ago. (See the JOURNAL, May, 1928, p. 2.)

The polls will remain open until March 12, 1929, and the result of the election will be announced in the April issue of the JOURNAL. Quite a few members in District No. 2 are depriving themselves of the privilege of voting in this election, through their failure to take care of their 1929 dues.

This is also the year for an Executive Board election in District No. 4, which is comprised of the following territory: Kentucky, West Virginia, Virginia, Maryland, District of Columbia, Tennessee, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Florida, Cuba and South America. Ballots asking for nominations for a member of the Board to represent District No. 4 will be mailed to all paid-up members in the District on February 13. If you have not paid your dues for 1929, do it now.

TO THE MEN

At the meeting of the Women's Auxiliary, last summer, some very interesting papers were presented and the women attending were greatly pleased with the program.

With the new officers bringing their enthusiasm into the work, coming meetings will be even more interesting.

Our organization is an exclusive one, as the membership is limited to the wives, daughters, mothers and sisters of the members of the American Veterinary Medical Association.

Is your wife a member? If not, she is missing a great opportunity. We have helped many students to finish their educations and complete their courses in veterinary medicine by lending them money at a low rate of interest. We hope to be able soon to offer a scholarship and thus further assist young men who are entering your profession.

Have your wife join the Women's Auxiliary to the American Veterinary Medical Association and help us attain our ambition.

MRS. CHARLES E. COTTON.

Recently a certain learned professor in an eastern university cast doubt on the authenticity of the famous letter written by Lincoln to Mrs. Bixby. Next somebody will try to tell the world that Paul Revere did not ride a horse, back in April of 1775.

APPLICATIONS FOR MEMBERSHIP

(See December, 1928, JOURNAL)

FIRST LISTING

- BERGAN, JOSEPH ARTHUR 2851 E. 5th St., Long Beach, Calif.
D. V. S., Kansas City Veterinary College, 1909
Vouchers: W. L. Curtis and C. C. Oderkirk.
- BOLLMEIER, WILLIAM THEODORE 641 N. 9th St., East St. Louis, Ill.
D. V. M., Ohio State University, 1918
Vouchers: A. E. Bott and H. R. Schwarze.
- BROWN, SYLVESTER 810 Hodley St., Whittier, Calif.
D. V. M., Chicago Veterinary College, 1912
Vouchers: W. L. Curtis and John L. Tyler.
- BRUNSON, ROBERT LEROY 918 Sheridan St., Corona, Calif.
D. V. M., Kansas City Veterinary College, 1914
Vouchers: P. H. Blickenstaff and W. L. Curtis.
- BUSHONG, REX DEAN 810 Houston St., Manhattan, Kans.
D. V. M., Kansas State Agricultural College, 1921
Vouchers: Edwin J. Erick and Edward R. Frank.
- CAMPBELL, HARVEY W. 43A So. Catalina St., Ventura, Calif.
D. V. M., Colorado Agricultural College, 1922
Vouchers: W. L. Curtis and J. P. Bushong.
- CLEMMER, HOMER R. Staunton, Va.
V. S., Ontario Veterinary College, 1906
Vouchers: Geo. C. Faville and H. C. Givens.

- DAVIDSON, WALTER W. 249 E. Jackson St., Stockton, Calif.
D. V. M., Washington State College, 1920
Vouchers: A. C. Rosenberger and J. P. Iverson.
- DELEZ, ARTHUR LOUIS University Farm, St. Paul, Minn.
D. V. M., Colorado Agricultural College, 1925
M. S., Michigan State College, 1928
Vouchers: R. Fenstermacher and H. C. H. Kernkamp.
- DUNLAP, GLEN LEROY Massachusetts Agricultural College, Amherst, Mass.
D. V. M., Kansas State Agricultural College, 1928
Vouchers: W. R. Hinshaw and E. F. Sanders.
- EDWARDS, FRANK 323 Chester Ave., Bakersfield, Calif.
D. V. S., Kansas City Veterinary College, 1909
Vouchers, W. L. Curtis and S. L. Edwards.
- ELBERSON, NOEL C. 15 E. 6th St., Anderson, Ind.
D. V. M., Terre Haute Veterinary College, 1912
Vouchers: R. H. Boyd and T. A. Sigler.
- FOLEY, TIMOTHY J. Frankfort, Kans.
D. V. M., Kansas State Agricultural College, 1923
Vouchers: B. W. Conrad and R. R. Dykstra.
- FOSBINDER, HARRY RISDON 1844 No. Berendo St., Hollywood, Calif.
D. V. M., Chicago Veterinary College, 1910
Vouchers: W. L. Curtis and L. M. Hurt.
- FUCHS, GEORGE ANDREW 125 Hackberry Ave., Modesto, Calif.
D. V. M., San Francisco Veterinary College, 1912
Vouchers: A. C. Rosenberger and J. P. Iverson.
- GOULD, HOMER A. 80 N. Daisy St., Pasadena, Calif.
D. V. M., Chicago Veterinary College, 1915
Vouchers: W. L. Curtis and T. H. Agnew.
- GRIFFITHS, C. B. 4226 E. Sacramento, Chico, Calif.
D. V. M., Kansas State Agricultural College, 1918
Vouchers: A. C. Rosenberger and I. G. LaRue.
- GRINER, ADLAI BEE Box 203, Fitzgerald, Ga.
D. V. M., University of Georgia, 1926
Vouchers: J. E. Severin and H. V. Persells.
- HEITT, JAY LEWIS 1235 Johnson St., Red Bluff, Calif.
D. V. M., Chicago Veterinary College, 1920
Vouchers: A. C. Rosenberger and W. L. Curtis.
- KENNEDY, GEORGE RALPH Box 183, Chase, Kans.
D. V. M., St. Joseph Veterinary College, 1918
Vouchers: C. M. Downing and B. W. Conrad.
- KEPPEL, JOHN 116 Center St., East Lansing, Mich.
D. V. M., McKILLIP Veterinary College, 1902
Vouchers: M. P. Hunt and T. S. Rich.
- KETCHAM, HAROLD FULLER Box 1337, Pawhuska, Okla.
D. V. M., Grand Rapids Veterinary College, 1909
Vouchers: C. R. Walter and F. F. Meads.
- LABAR, CHAUNCEY F. 606 Congress St., Ypsilanti, Mich.
D. V. M., Michigan State College, 1919
Vouchers: B. J. Killham and Ward Giltner.
- LITTLE, HAROLD F. St. George Hotel, Santa Cruz, Calif.
D. V. M., Indiana Veterinary College, 1921
Vouchers: A. C. Rosenberger and I. G. LaRue.
- MCCRILLIS, JOHN J. 28 Valley St., Pasadena, Calif.
D. V. M., Ohio State University, 1923
Vouchers: W. L. Curtis and F. P. Wilcox.
- MCGARRY, JOHN A. 158 Front St., Santa Cruz, Calif.
M. D. C., Chicago Veterinary College, 1896
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- MCGARRY, JOHN A. 158 Front St., Santa Cruz, Calif.
M. D. C., Chicago Veterinary College, 1896
Vouchers: W. E. Curtis and J. P. Bushong.

- PARRISH, R. D. Porterville, Calif.
D. V. M., Kansas State Agricultural College, 1914
Vouchers: L. O. Henrich and W. L. Curtis.
- PROPER, C. F. Box 1394, c/o P. M. Dairy, San Diego, Calif.
D. V. M., Chicago Veterinary College, 1911
Vouchers: W. L. Curtis and E. P. Wilcox.
- RAMSEY, ROY L. 114 Monroe St., Lapeer, Mich.
D. V. S., Grand Rapids Veterinary College, 1906
Vouchers: M. P. Hunt and B. J. Killham.
- REID, FRANCIS JOSEPH St. Marys, Kans.
D. V. M., St. Joseph Veterinary College, 1918
Vouchers: L. P. Gentry and W. B. Tipton.
- ROBB, JOSEPH RALPH 888 Home Ave., Oak Park, Ill.
D. V. M., Ohio State University, 1928
Vouchers: O. V. Brumley and J. V. Lacroix.
- ROOT, ROBERT R. Tres Pinos, Calif.
D. V. M., San Francisco Veterinary College, 1915
Vouchers: A. C. Rosenberger and F. H. McNair.
- SCHATTENBURG, AUGUST E. Riley, Kans.
D. V. M., Kansas State Agricultural College, 1919
Vouchers: James F. Adece and J. F. Hemphill.
- SCHMIDT, ALBERT IRVING Box 367, Cos Cob, Conn.
D. V. M., Kansas State Agricultural College, 1928
Vouchers: Wallace F. Vail and George L. Cheney.
- SEABURY, WILLIAM AUGUSTUS 818 North D St., Madera, Calif.
D. V. S., Grand Rapids Veterinary College, 1899
Vouchers: A. C. Rosenberger and Fred L. O'Neal.
- SETTLE, DAVID EARL 405 S. Butte St., Willows, Calif.
D. V. S., San Francisco Veterinary College, 1907
Vouchers: David F. Fox and A. C. Rosenberger.
- SPURLOCK, JACK HARVEY State Office Bldg., Trenton, N. J.
D. V. M., Kansas State Agricultural College, 1928
Vouchers: J. H. McNeil and Henry H. Haigh.
- STEVENS, CHAUNCEY C. 1218 Minnie St., Port Huron, Mich.
V. S., Ontario Veterinary College, 1906
Vouchers: H. Preston Hoskins and B. J. Killham.
- TIMMIS, R. W. 1537 East 12th St., Oakland, Calif.
D. V. M., Chicago Veterinary College, 1920
Vouchers: W. L. Curtis and Joseph M. Arburua.
- TUTTLE, LEWIS EDMUND Willows, Calif.
D. V. S., San Francisco Veterinary College, 1907
Vouchers: W. L. Burtis and J. P. Bushong.
- WALTER, CHARLES P. O. Box 846, Zionsville, Ind.
D. V. M., Terre Haute Veterinary College, 1915
Vouchers: Robert Thumann and Benj. H. Yenner.
- WATKINS, S. E. Hanford, Calif.
D. V. S., Kansas City Veterinary College, 1904
Vouchers: L. O. Henrich and C. R. Rey.
- WIMSETT, IRA G. Wellington, Kans.
V. S., Indiana Veterinary College, 1902
Vouchers: J. H. Burt and L. A. Hammers.
- WINTER, ASA Devils Lake, Mich.
D. V. M., Michigan State College, 1921
Vouchers: M. P. Hunt and T. S. Rich.
- WINTER, JOHN H. 130 Toledo St., Adrian, Mich.
D. V. M., Grand Rapids Veterinary College, 1917
Vouchers: M. P. Hunt and B. J. Killham.

Applications Pending

SECOND LISTING

Ahr, Daniel M., c/o Bellevue Ranch, Merced, Calif.
Alexander, Oliver C., 1123 1/3 S. 7th St., Springfield, Ill.
Baxter, Joseph M., 4823 N. Sornock St., Philadelphia, Pa.
Bernhardt, Ralph Walter, Enderlin, N. Dak.
Campbell, Ora L., Box 308, Astoria, Ill.
Cilker, Robert Abner, 1414 9th St., Modesto, Calif.
Crossland, Ralph Eldred, Neponset, Ill.
Curran, Robert F., Buda, Ill.
Dickie, Samuel R., Paw Paw, Ill.
Duckworth, R. E., 828 Euclid Ave., Berkeley, Calif.
Erickson, Arthur John, Toulon, Ill.
Feers, Albert Gurney, 4140 Second Ave., Los Angeles, Calif.
Garrett, George Matlack, 701 S. Walnut St., West Chester, Pa.
Gray, G. A., Adair, Ill.
Griesemer, Samuel F., R. F. D. No. 1, Temple, Pa.
Grossman, Harry T., 2237 W. Grand Ave., Detroit, Mich.
Hackler, Henry D., 2055 E. Susquehanna Ave., Philadelphia, Pa.
Hannum, Henry B., Brandywine Summit, Pa.
Hawes, John Everett, 1000 West Yosemite Ave., Madera, Calif.
Hope, Fred S., 148 N. Paxon St., Philadelphia, Pa.
Hornbaker, H. R., 759 E. Main St., Galesburg, Ill.
Hughes, E. C., Carlinville, Ill.
Jones, Kenneth Uttley, 901 Second Ave., N., Birmingham, Ala.
Lair, M. T., Alexis, Ill.
Leahy, Glenn W., R. 5, Decatur, Ill.
Legner, Ernest F., 519 N. Hennepin Ave., Dixon, Ill.
Maloney, Michael John, 5423 Christian St., Philadelphia, Pa.
Marquiss, Floyd D., 1443 Stannage Ave., Berkeley, Calif.
O'Rurke, Michael John, 720 Valencia St., San Francisco, Calif.
Puckett, L. V., 1136 N. 20th St., Quincy, Ill.
Rockwell, C. S., 5225 Spruce St., Philadelphia, Pa.
Rothermel, Robert O., 550 N. Fifth St., Reading, Pa.
Sheets, Charles F., Delphi, Ind.
Shepherd, Fred Allen, 324 Allen St., Belvidere, Ill.
Steiger, Clarence Melville, Route A, Box 38, Modesto, Calif.
Swanson, Andrew, Atkinson, Ill.
Thomas, William W., P. O. Box 563, Merced, Calif.
Turner, John, 1241 E. 12th St., Oakland, Calif.
Whitaker, Andrew J., Manteca, Calif.
Whitehead, Frank George, 923 St. Helena Ave., Santa Rosa, Calif.
Yowell, Daniel A., Virginia, Ill.
Zollinger, John H., 1 Chelton Rd., Llanerch, Upper Darby, Pa.

The amount which shall accompany an application this month is \$9.58, which covers membership fee and dues to January 1, 1930, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Alabama Veterinary Medical Association and Short Course for
Graduate Veterinarians. Auburn, Ala. February 4-9, 1929.
Dr. C. A. Cary, Secretary, Auburn, Ala.

Connecticut Veterinary Medical Association. Hotel Garde,
Hartford, Conn. February 6, 1929. Dr. E. H. Patchen,
Secretary, Milford, Conn.

- New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. & 103rd St., New York, N. Y. February 6, 1929. Dr. Raymond J. Garbutt Secretary, Avenue A and 24th St., New York City, N. Y.
- San Diego-Imperial Veterinary Medical Association. San Diego, Calif. February 6, 1929. Dr. A. P. Immenschuh, Secretary, Santee, Calif.
- Kansas State Agricultural College Conference for Veterinarians. Kansas State Agricultural College, Manhattan, Kans. February 6-7, 1929. Dr. R. R. Dykstra, Kansas State Agricultural College, Manhattan, Kans.
- Illinois Veterinary Conference, University of. Urbana, Ill. February 11-13, 1929. Dr. Robert Graham, Secretary, University of Illinois, Urbana, Ill.
- Chicago Veterinary Society. Great Northern Hotel, Chicago, Ill. February 12, 1929. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.
- Hudson Valley Veterinary Medical Society. Albany, N. Y. February 13, 1929. Dr. J. G. Wills, Secretary, 122 State St., Albany, N. Y.
- Southeastern States Veterinary Medical Association. Hotel Patton, Chattanooga, Tenn. February 15-16, 1929. Dr. M. R. Blackstock, Secretary, 157 Hampton Ave., Spartanburg, S. C.
- Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo. February 19, 1929. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.
- Ontario Veterinary Association. Prince George Hotel, Toronto, Ont. February 20, 1929. Dr. H. M. LeGard, Secretary, 223 Main St. N., Weston, Ont.
- Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. February 20, 1929. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.
- Northwestern Ohio Veterinary Medical Association. Commodore Perry Hotel, Toledo, Ohio. February 21, 1929. Dr. F. A. Lambert, Secretary, c/o the Columbus Serum Company, Station G., Columbus, Ohio.
- Ohio State University Conference of Veterinarians. Columbus, Ohio. March 20-22, 1929. Dr. D. S. White, Dean, Ohio State University, Columbus, Ohio.

RESULTS OF RESEARCH ON DISEASES OF FUR-BEARING ANIMALS IN CAPTIVITY*

By R. G. GREEN,

*Department of Bacteriology and Immunology, University of
Minnesota, Minneapolis, Minn.,*

and J. E. SHILLINGER,

*Bureau of Biological Survey, U. S. Department of Agriculture,
Washington, D. C.*

The cooperative research on diseases of fur-bearing animals, being carried on jointly by the University of Minnesota and the Bureau of Biological Survey, had its inception in the fall of 1927, both institutions having carried on independent investigations prior to that time. The University group began its work on this subject in January, 1925, and carried out a program of experimental investigations continuously until it combined its researches with the Government Bureau. The Bureau of Biological Survey began its work on this subject in 1921, and has carried on numerous field investigations of epizootic diseases occurring on ranches raising fur-bearing animals in captivity. With the merging of these two investigations, it has been possible to carry out coordinately both field work and experimental investigation with thoroughness on an adequate scale.

Thus far investigations have been directed to the studies of epizootic diseases occurring on fox ranches. In a sense the work has consisted of an investigation of so-called distemper in foxes. Previous to this work the term "fox distemper" had come into usage, because of the occurrence of a disease or diseases in foxes similar in their manifestation to dog distemper. There appeared forms of disease in foxes which could be ascribed as the upper respiratory, the intestinal, and the central nervous system types of distemper. It has been considered from the outset, however, that foxes must be susceptible to more than one disease, and that the term "distemper" in its usage might be applied, in the state of incomplete knowledge, to one or more of several diseases. In our investigations up to the present time we have not attempted to define any disease of foxes as a disease to be called distemper, but have simply carried out extensive and complete studies of

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any infectious condition occurring on a fox farm in such proportions as necessitate its being considered an epizootic disease.

The general plan of the investigation has been to confine study to a single epizootic in progress, to gather adequate information concerning the disease in ranch animals, and then to transmit the disease to healthy foxes, and study the disease in its entire course in such animals kept under close observation. The study of each ranch epizootic has included the duration of an epizootic as a whole, the duration of disease in individual animals, the symptoms exhibited, complete postmortem findings, extensive cultural studies of all organs, and a study of microscopic pathology of all organs including the central nervous system. Attempts were made to find what organs from a ranch animal would be infective, and this material studied very carefully and completely to determine the nature of the infectious agent. Animals in which the disease was artificially produced were studied as completely as the ranch animals.

It would appear that the work thus far carried out along these lines has given us a definite insight into two epizootic diseases that may occur in fox ranches. Both of these diseases are recognized as entities by the investigators, and at present are referred to under the names of "fox paratyphoid" and "epizootic fox encephalitis."

FOX PARATYPHOID

The disease that we have called fox paratyphoid¹ was prevalent in various parts of the country prior to and especially during the year 1925. It appeared to be more prevalent among young fox pups than it did among the adults. As an example, on one ranch studied, the proportion of deaths was approximately ten fox pups to one adult. The disease occurred in an epizootic, which appeared to be an uncomplicated fox paratyphoid. The mortality rate was approximately 60 per cent of the puppies born that year. The disease appeared to be of considerable duration in the individual animals, as there was an appreciable loss in their weight. In experiment animals the course of a fatal infection appeared to be about eighteen days. The eyes and nose do not present a noticeable purulent discharge. At death, however, the eyes are markedly sunken, the cornea often being entirely inside the bony structure. The symptoms are loss of weight and weakness, an occasional fox dying with convulsions. Postmortem findings show a normal upper respiratory tract, pneumonia being of rare

occurrence. The intestinal tract frequently shows marked inflammation; the spleen is usually enlarged and dark.

From animals dying on the ranch an organism belonging to the paratyphoid group could be isolated in pure culture. When this organism was injected into healthy foxes it produced a disease similar to that seen in animals dying under ranch conditions, and the postmortem findings were identical. The organism could be passed serially from fox to fox, producing the typical infection in each animal.

The picture described above is that observed on a ranch where the disease was present during the summer. An epizootic disease that appears to be fundamentally the same was studied during the winter months. In these outbreaks a respiratory involvement was an addition to the former picture. In these cases, however, the paratyphoid organism could be demonstrated in the ranch animals and was isolated in pure culture. Injection into healthy animals produced a fatal disease in which, however, the upper respiratory symptoms and pneumonia were lacking. It has been our opinion that this type of epizootic also represents in fact a paratyphoid infection, and that a secondary infectious agent responsible for the respiratory involvement was passed from animal to animal along with the primary typhoid disease under ranch conditions but was lost when the transmission was made under laboratory conditions.

Very careful consideration has been given to the question of the paratyphoid organism, being in itself a secondary invader. While it is possible that a filtrable virus might accompany this organism, such could not be demonstrated. Very strong evidence that the paratyphoid organism was *the cause of this disease* has been gained from our experience since that time. Although numerous epizootics were studied during the years 1926 and 1927, the paratyphoid organism was not met with as an accidental, secondary invader.

FOX ENCEPHALITIS

The disease that we have called epizootic fox encephalitis^{2,3} was the second disease of foxes recognized by the group of workers at the University of Minnesota. Up to the present time the co-operative research has been directed to gain a more complete understanding of this disease. This epizootic appears to have been widespread during the years 1926 and 1927. It was known to have been introduced on a number of farms in several states by foxes that had been exhibited at fox shows. This disease has

been in evidence also during the present year. Because it is very insidious in its manifestation and has a fairly low mortality rate, it was recognized as an epizootic disease in many cases only with difficulty. The infection spreads quite slowly under ranch conditions, but becomes plainly an acute epizootic disease among foxes herded together in a fur-yard.

The disease appears to attack both young and adults in about the same proportion. The mortality rate is usually not over 15 per cent of the ranch population, but occasionally may be as high as 40 per cent. Unlike the paratyphoid infection, a fatal termination is liable to result very early in the course of the disease. For that reason the foxes dying are usually in good flesh. The disease itself may be of several weeks duration, but it appears that any fatal termination regularly occurs within the first two weeks and frequently in much less time.

Most animals dying during an outbreak will be found dead, and usually it will be reported that each fox was seen some hours before and appeared to be perfectly healthy. The basis for this sudden death will be apparent from the pathology described later. In uncomplicated cases of this infection the eyes and nose do not present a purulent discharge and at death the eyes are not sunken. Symptoms of this disease under ranch conditions are very difficult to get because death is usually quite sudden.

When symptoms are observed on ranches, they are generally nervous manifestations. A series of convulsions is the most common symptom. An apparent sleeping or lethargic state is quite characteristic of the disease and occurs in many cases. A sudden, extreme weakness is frequent. This is often difficult to differentiate from paralysis of many groups of muscles. Paralysis may occur, however, involving various muscle groups and may be either spastic or flaccid. Death is usually in coma. Postmortem findings of this disease have been most baffling and macroscopic examination of foxes dying of this infection is apt to reveal no sign of any anatomical change. The various organs may appear to be perfectly normal. In most cases there are hemorrhages of varying extent found in the internal organs. These hemorrhages occasionally are very marked but usually appear almost insignificant. They appear as subpericardial and subendocardial hemorrhages, as irregular hemorrhages in the thymus, adrenals, pancreas, and submaxillary glands. Spots of hemorrhages may occur in the lungs or digestive tract. Sometimes there are very marked gross hemorrhages in the pleural and peritoneal cavities,

and in the lumen of the stomach and intestines. Occasionally a gross hemorrhage is met with in the brain and spinal cord, and it is this finding that gives the clue to the most important microscopic lesions found in this disease.

Microscopic examination of the brain and spinal cord shows that marked changes occur in the central nervous system. These changes are represented by perivascular infiltration of cells around the blood-vessels, by clumps and nests of round cells, and by hemorrhages in the nervous tissues. These lesions appear to be much more common in the gray matter and especially in the region of the medulla.

It has been found that the disease can be transmitted from sick animals to healthy animals by the injection of brain material derived from an animal suffering from the disease. This infectious material appears to give most consistent results when injected directly into a silent area of the brain or introduced into the cisterna magna. Animals receiving this injection show no ill effects immediately after the operation, appearing perfectly normal after recovering from the effects of the ether. Symptoms of the disease occur usually in from four to seven days, and death usually results within twelve hours after the first symptoms. The symptoms are identical, both in their manifestation and variability, with those observed in animals dying in a ranch epizootic. The disease may be transmitted in series from one fox to another. The postmortem findings appear to be identical with those found in ranch animals and the microscopic pathology is the same.

Epizootic fox encephalitis may be a generalized infection. The nature of the lesions in the central nervous system, however, appears to make this aspect of the disease most important, as the grave symptoms produced and the fatal termination appear to be usually associated with the changes taking place in the central nervous system. For this reason we have considered that this disease should be referred to as an encephalitis.

Tests that were far from conclusive have been made in certain other fur-bearing animals and dogs to determine the infectivity of this disease for them. Dunkin and Laidlow⁴ found the strain of virus causing dog distemper with which they were working readily transmissible from dogs to ferrets and back again to dogs. The causative agent of epizootic fox encephalitis appears to be non-infective to ferrets. It might be responsible for some infections in dogs and minks and may be carried by them, but this possibility must receive much more extensive study before con-

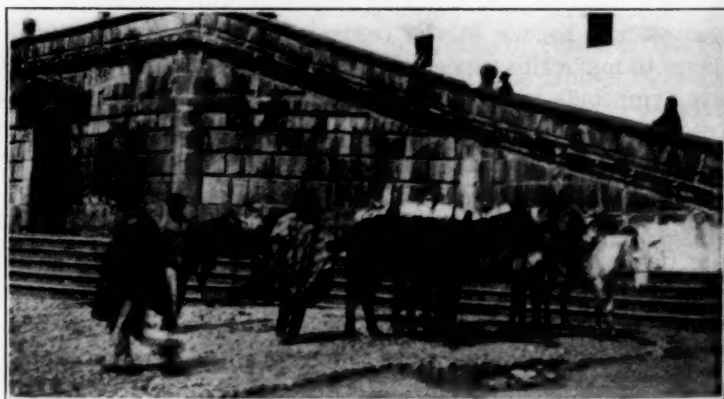
clusions can be established. Certain symptoms of this disease simulate those recognized in distemper of dogs very closely, but work done up to the present indicates that the two diseases are distinct.

SUMMARY

Two epizootic diseases thus far have been described for foxes—one an infection by a species of paratyphoid bacillus, and the other an encephalitis that does not appear to be caused by a bacterium, but rather by a filtrable virus. The paratyphoid infection is a disease more highly contagious for young animals, having a duration of two weeks or more and characterized by gross changes in the intestinal tract, spleen, and other internal organs. Epizootic fox encephalitis is a disease of both young and adult animals, fatal termination resulting usually after a short interval of the disease, and is characterized by lack of marked gross pathology, the pathology consisting mainly of microscopic changes in the central nervous system. The mortality rate of the paratyphoid infection is usually over 50 per cent. The mortality rate of fox encephalitis is usually between 10 and 20 per cent but may be as high as 40 per cent of the animals on a ranch.

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A MILKMAN OF QUITO, ECUADOR

The milkman has just dismounted and is starting to unload the cans from the backs of his burros. Picture taken by Lieutenant Colonel Robert J. Foster, V. C., U. S. A., on a visit to Ecuador in November.

THE ONLY SOURCE OF SUPPLY OF THE PROFESSION*

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The things I touch upon briefly in this paper might be greatly elaborated upon if more time were available. I hope, however, in the time allotted, to appeal to the profession for a greater interest in, a greater helpfulness to, and a greater cooperation with the profession's only source of supply—the school. I may not speak with great authority on this subject, but I certainly can from long experience. For thirty-five years I have been a veterinary pedagog and for thirty-two years a combination of pedagog and administrator.

We commonly speak of ourselves as belonging to the "veterinary profession." What makes the group of men with whom we classify ourselves a profession? What is a profession, anyway? Webster defines it as "a calling in which one professes to have acquired some special knowledge used by way either of instructing, guiding, or advising others or serving them in some art." To the average person a profession is a *group of people*, who pronounce publicly the possession of expert knowledge gained through the school, supplemented by practical experience. Some of the older groups, like law, medicine and divinity, are spoken of as the "learned" professions, implying learning, erudition, scholarship, all of which hark back to the school. As yet, we have not been grouped among the learned professions. This is largely on account of ignorance, and the too common habit of thought which seems to link us closely with agriculture. While to be sure the veterinary profession is an aid to agriculture, it is no more so than any other of the professions. If linked up with anything, it should be with medicine, the profession which deals with diseases in the higher animal we call man.

Fundamentally it differs little whether the inflammatory process be located in an old senator's big toe or an old cow's ear; it is the same series of phenomena, the same resistance of the cells to the attack of the irritant upon some part of the living body. However, dentistry (a branch of human surgery) is not yet among the "learned" either; pharmacy, now so vitiated with

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commercialism, is probably farther away from it today than it was twenty-five years ago. In this age we find the route to the profession always through the school. Even in law fewer boys are found "reading" in some attorney's office and more of them in the law schools of the country. In the production of the professional man, the school stands out as the most important link in the chain of education, training and experience which leads from ignorance into the realm of the learned.

Were we to abolish professional schools, the professions would die. While formerly each profession attempted to support its own schools, today professional schools are included in most of our land grant colleges, receiving direct support from the state and thus becoming incorporated in the public educational system of the state. Where a professional veterinary course is offered by a state university along side of law, medicine and engineering, the effect upon the people of the state is good for the veterinary profession. Because of the very fact that several thousand students come into more or less direct contact with the veterinary school, an immeasurable influence for our good is exerted, provided the school is organized, manned and equipped on a parity with the other university divisions. In considering the school I shall touch upon the entrance requirements, student body, faculty, buildings and equipment and do so very briefly.

ENTRANCE REQUIREMENTS

Up until ten years ago, the entrance requirements to our schools were placed at a point permitting the greatest number to matriculate and still maintain some sort of a standard. In some instances, only the common school education was required; in others, one or two years of high school. Since the war, all have made high school graduation or its academic equivalent the minimal requirement. In the meantime the "learned" professions moved up several notches beyond this, medicine requiring two to four years of college training and dentistry one to two years. This change brought them nearer to European conditions governing the entrance to a profession, and gave the schools a more mature group of students, young people with a better knowledge of the fundamentals of science, language, history, mathematics and the humanities and thus much better prepared to cope with the professional curriculum. It also permitted the school to raise its standards of study from, in many instances,

the introductory consideration of a subject to the more profound and exhaustive consideration.

A student who studied zoology and comparative anatomy in an arts college, finds handling terminology, the freshman bugbear, much easier. Thus, with a better educated, more mature student, a better course in a subject could be given, and given more rapidly, covering much more ground in each course pursued. The net result is that the product of the school is a more mature, better educated and better trained individual, competent to enter professional life with a broader viewpoint, greater confidence, more self-esteem and a better approach in meeting up with people. The confidence of the thinking laity in the medical profession was at a much lower ebb twenty-five years ago than it is today. There are more educated people abroad than ever before in the history of the world, especially in the United States, when our educational facilities are taxed almost to the breaking point. I have seen the population of a single university grow from 150 to 15,000 students.

The product of our veterinary schools must make contacts with more educated laymen and should be able to meet them on a footing equal to that of their physicians, dentists and lawyers. It is significant when 85 per cent of the graduating class in medicine hold baccalaureate degrees, while less than 2 per cent of the veterinary graduates on the same platform can boast of any preliminary education beyond the high school! I fully realize that there are reasons for this. One reason is that we have too many poorly equipped veterinary schools to create an atmosphere of respect and confidence from the college-educated man. Another reason is the great difference in the size of our profession in comparison with the other professions. There are twice as many students in the medical schools of the United States as there are graduate veterinarians in the whole country. A third reason is the belief that the emoluments of our profession suffer when comparisons are made with the other professions. I think the average student takes Emerson's advice and "hitches his wagon to a star." He is fully convinced that if a great surgeon can collect in fees \$100,000 in a year, his income, once he is going, will probably exceed it. His eye is always fixed on the maximum. His expectations never include the minimum or even the average. In our profession he sees no such goal, and, already possessing a much higher education than we demand to enter the portals of our profession, passes us up.

We must not lose sight of the fact that the earning power of the individual is set by his limitations. A man worth only \$100 a month will in the long run reach about that level no matter where you place him, unless, by some artificial means, you can make a monopolist of him. While I firmly believe in laws regulating professional practice and all my life have fought to create them, I doubt very much their effect upon the income of the really competent, who can render real service. They are always most helpful to the weak sister among us.

We can not continue to take the rejects, the culls, the boys who wanted to study something "higher" and failed and with this material build up a great veterinary profession. Medicine is forging far ahead of us because she is in a position to use only the best material out of which to make physicians. In one university medical school not over 50 per cent of those applying for entrance are admitted. Classes are limited to seventy-five students each, and every effort is made to permit only the fittest to survive the strenuous curriculum. Just the opposite conditions prevail in the veterinary school of the same university.

THE SCHOOLS

Time will not permit of a discussion of our schools. In passing, however, it may be remarked that more than half of them are inadequately housed, equipped or staffed. Compared with the schools in continental Europe, even the best of them fail to meet the minimum standards of efficiency. When we compare the relative wealth of America and the value of her live stock industry with that of any European country, the money our states have put into veterinary schools is miserably small. But we began badly. Patterning after "Mother" England, we tried to make our professional schools self-supporting. State support is of very recent origin. To make the school pay, demanded that the entrance requirement be kept low to encourage a large student body, the faculty paid very little, if anything, except in the prestige from being associated with a college, very useful in dealing with the public through practice. For financial reasons the housing facilities and equipment were kept at the lowest possible level.

In 1890, when I went abroad to study, there was a vacant lot in the city in which I lived. When I returned, three years later, there stood on this lot a medical university with over 400 students which had already turned out one class. Obviously, little

beyond the art of practice could be taught under the conditions prevailing. There was neither time nor talent for the fundamentals which were looked upon as merely theory, just as if anything is possible of accomplishment without theory and the application of fundamental principles whether or not they be known or understood by the artist.

In the past quarter of a century the learned professions have learned much. The better medical school of today is either supported by the state or possesses a generous endowment; the entrance requirements, as noted, assume a prescribed college education; the faculty is becoming more and more full-time employees of the school; the buildings, including the hospitals, palatial and the equipment generous. Some of our modern medical schools represent a money investment running into the millions of dollars. The product is obviously capable of a much more efficient service to humanity through the relief it affords the suffering and the longer stretch of years it gives to the average human life.

In our profession, in part due to the lethargy and sometimes hostile spirit manifested toward increasing the standards of education and the development of the schools, we have failed to guard adequately the portals of the profession, with the result that too many of the mediocrity and too few of the superiority are filling in the gaps in the profession caused by the ravage of time.

I believe the time has come for a decided change. The attitude of the average, middle-of-the-road member of the profession must turn from that of mere expediency in this matter to something more fundamental. To protect himself he must look beyond himself. Too many of us have allowed ourselves to become "short-headed" in the matter of the school. Too often have I heard a veterinarian burst into caustic criticism of his alma mater, declaring it to be the poorest school on earth, and then refuse to do anything constructive to aid her in becoming the greatest school on earth.

There are in the United States eleven recognized veterinary schools. All but one is either an integral part of or closely affiliated with a land grant university. In these eleven schools, last November, were 668 students, or an average of 61 to each school. The matriculation varied from nine students in the smallest school to 114 in the largest. In attendance, the four largest schools are New York, Iowa, Ohio and Kansas; the four

smallest, Texas, Georgia, Alabama and Michigan. Veterinary education does not seem popular in the southern states. Their largest school has only eighteen students. Generally speaking, the school attendance goes hand in hand with the value of the live stock industry of the state. There are, however, other factors. Probably Michigan's close proximity to Ohio, New York and Pennsylvania, each equipped with older and richer schools, accounts for their lower enrollment. The greatest source of student supply is the loyal alumnus. Michigan began only nineteen years ago, while each of her competitors is over thirty years old and each has passed through the period of peak in veterinary student numbers and, therefore, large graduating classes—a large alumni body.

Many thinking veterinarians believe we have too many schools. Some have suggested that the number be reduced to three, each properly located geographically. Probably there are too many. This is a delicate question and one difficult to answer. Germany, about the size of Texas, with not one-third our animal wealth and with less than one-half our population, has five well-equipped and well-supported veterinary schools. France, with less than one-third our population and one-fourth our animal wealth, has three schools, and Great Britain, with 40,000,000 people and a live stock industry of less than one-quarter of ours, has four.

In brief, the total live stock numbers of Great Britain, France and Germany combined fall considerably below those of the United States. These three countries together possess one-half as many horses, two-thirds as many cattle, three-fifths as many swine and about the same number of sheep. To educate veterinarians to guard the health of animals in the combined countries are twelve schools with a student population of approximately double that of the United States. It is notable that many of our schools register only a few students each.

It is probable that the location of our schools, rather than the number, is at fault. If all of the students in all of the southern schools were placed in one school, the population of that one school still would be only two-thirds of the average population of all the schools.

But student population alone should never be the criterion to determine the existence of a school. Besides teaching and research, each veterinary school has a third function. It should head up, be the source of authoritative information, the court of last

resort in all matters relating to veterinary medicine other than regulatory. Notwithstanding a small matriculation, a school, therefore, can be a great asset to the commonwealth. Think of Sir Arnold Theiler's school in South Africa, where routine pedagogy was made secondary to research. It never mustered over twenty-five or thirty well-trained students, all that were needed to meet the demand of the state.

On one point, however, I do feel secure. I do not believe we need any more veterinary schools. "What we need in the United States is a sufficient number of schools of high class and not an excessive number of schools of low class." This sentence is quoted from a report made by the late Leonard Pearson, chairman of the Committee on Intelligence and Education, at the Kansas City meeting of the A. V. M. A., twenty-one years ago. At that time there were twice as many schools in existence, with three times the number of students, as there are today. Nevertheless, the statement of Dr. Pearson stands out as a beacon light issuing a warning against all effort to cheapen veterinary education. He also realized that any argument for progress in veterinary education is often met with the statement that one should not expect too much, for "the veterinary sciences are young in this country." But science knows no geographical or political boundaries. It is world-wide always. A scientific discovery made at dusk in Japan is known the following dawn all over the civilized world. It is not that our profession is so young (its methods are as old here as anywhere) but that it has not yet been given the fullest appreciation by our people, who, as noted, are not a live stock people in the old-country sense.

TEACHING STAFFS

It has been said that there are only a sufficient number of fully equipped teachers available to man one good school in the United States. Probably here again exaggeration was employed to emphasize the fact that there exists a shortage of men in our profession trained to become teachers and research workers in our veterinary schools. A greater number of young graduates should work for higher degrees. We are shy on Ph. D's. In our experience it is most difficult to influence the best material to continue their schooling for one to three years, studying to become specialists in some field in order later to become professors. Too often the least adapted, the fellow who fears to face the world on his own, and the poorer scholar is the one who thus prepares

himself. While formerly the teacher must have been a man with a good share of the spirit of the missionary who served for little pay, today the universities pay the highest salaries to outstanding teachers, making no distinction as to field except as the law of supply and demand be of influence. In Europe large numbers of young veterinarians are doing postgraduate work preparatory to accepting teaching positions. Through scholarship or fellowships, postgraduate training should be encouraged.

In what has gone before I have tried to sketch the situation as it concerns our schools, with a hint as to what must be done to bring them again to a par level. I have done so hoping to stimulate a greater and more intimate interest in the problem in the veterinary graduate. His present attitude is too much that of the tourist who has passed over a bad detour. He would like to forget it. But we can not forget our schools and continue as a flourishing profession. Just because the state has assumed the responsibility for their support does not mean that the profession can leave the matter entirely to the state. Veterinary medicine is only one of many phases of education which the state supports and it is the smallest of them all. Without constant manifest interest, the impersonal state, usually much tainted with politics, may either drop it from its list of beneficiaries, or continue it on in a spirit of mere tolerance rather than of promotion.

The veterinarian, therefore, who desires to see his profession continue to grow in the appreciation of the people, must become more alert to needs of the school and use his influence as a citizen and voter to further them. Among other things he should look forward to an increase in the entrance requirements in order to fill up the schools with a better educated, more mature type of student, whose intellectual plane is higher than at present and more on a par with the student in the other professions. He should be careful to recommend prospective students only when he knows them to be fit. More than once some loyal alumnus has introduced to me a prospective student whose only recommendation was that he had made a failure of everything else. Laymen have done the same too, even intelligent laymen.

An eminent divine of my home city, who has a number of sons, most of them in the profession and all except one doing well, tried to enter his less fortunate son in the veterinary school, with the confession that he tried him at everything else, even to driving a poll-wagon for a circus and he had failed. Kind deans of other colleges have frequently come to me with similar offerings in the

way of faltering students whom they wish off their hands. "This boy likes horses—he ought to make a great veterinarian." How often have we heard that. Either send us young men who can make good both in the school and, after graduation, out of it, or do not recommend at all. The school to the boy is what the training-track is to the race-horse. Neither can create something out of nothing. Ford's millions could not have made my faithful old buggy horse of twenty years ago trot a mile in four minutes. The boy who has no mentality, who can not make good in any school, will not make good in veterinary medicine. He may become a useful, good citizen and die respected by his fellow man but he will never do in a "learned" profession.

When the veterinary school is asking for greater support from the legislature, the intelligent veterinarian will realize that what helps the school, helps the whole profession and this helps him, and will use his influence with the legislature in the matter. As a voting outsider his influence is much greater with the average legislator than that of a university president or a dean, looked upon mainly as self-interested parties in the appeal. Other professions do these things; why not we? In one state a veterinary graduate in the legislature did everything he could to embarrass the school of veterinary medicine at the land grant university, even to the point of trying to have it abolished by law. I have not found that members of our profession in legislative bodies have been more useful in promoting veterinary education than laymen, and some have proven more of a liability than an asset in securing increased appropriations for the school.

There are, of course, many notable exceptions, and when a strong man from among us enters the halls of legislation, veterinary laws and schools ultimately feel the effect of it. No one knows more than I of our progress in the past third of a century. We had a long, long trail ahead, 'way back there in the "good old days" of the livery stable, odors of horse manure often commingling with iodoform, each odor struggling for supremacy; the weekly shave, the "chaw" of "Mail Pouch," the noise of blatant self-advertising, and the venomous criticism of the "opposition." The younger element know nothing of this. To some people, to be dirty meant to be practicable—some of our fore-runners were eminently practical as judged by that standard. In those days the private school taught only the *art* of veterinary medicine, and the few state schools less of the art and little of the science.

Since then we have made progress undoubtedly—but has not most of the progress been outside of the school? Is there not a danger that the profession will outstrip the school? The veterinarian's education *begins* on commencement day. It does not *stop* there.

Does the American veterinary college today bear the same relationship, the same spirit of leadership, that the European school enjoys in this regard? Do our professors attain the same high standing and respect here as they do abroad? I do not think so. But they should and it is up to the profession to make this demand. The voice of this association should be heard in the whole matter of placing each school we recognize in the position of educational leadership it should enjoy. That is your problem just as much as it is the problem of the school itself. Your interest and advice is, therefore, earnestly solicited. Outside pressure has proven much more powerful than inside in dealing with higher university authorities and with legislatures. I would rather have the earnest, well-directed work of one veterinarian, a voter and tax-payer, than the united force of the whole veterinary faculty, handicapped as it sometimes is by internal dissensions, petty politics and seemingly selfish desires. We must keep in mind the fact that the higher educational authorities and legislative bodies concerned are not particularly interested in our field of endeavor, and to influence them requires the outside factor—the group of people in the state, who vote, and are in a position to demand.

As the only source of supply of the profession, the portal through which each one of our new members must pass, stands the school. To keep it at par, to build it up, to guard it with solicitude in order that only the best may pass out of it and into our ranks is the job not only of the powers of education at large, or of the dean and faculties, but of the whole profession.

Let our only source of supply so function that each succeeding year the gaps in our ranks are filled with men of better education, better training, better prepared to render service, and better able to stand in favorable comparison with the output in the other "learned" professions.

THE SHIFTING AGE

1900—"Do not hitch horses to the trees."

1929—"No parking on this side."

ESSENTIALS IN VETERINARY EDUCATION*

By GEO. H. GLOVER, *Fort Collins, Colo.*

Dean, Division of Veterinary Medicine, Colorado Agricultural College

Surely not much can be expected of a twenty-minute paper dealing with the very broad subject of veterinary education. I shall treat it in a very general way and can only hope that something said will stimulate a worthwhile discussion.

We have gathered here in the spirit of reciprocity, for we are in accord that no one is sufficient unto himself alone. We have come from nearly, if not every, state in the Union, from the provinces of Canada, and have a few visitors from foreign countries. This being the Section on Education and Research, it is assumed that probably all the veterinary colleges are represented. This association has been the alma mater to the veterinary profession and as such has wisely directed its educational and ethical trend.

Every merchant, who knows what he is doing, takes stock annually and adjusts his business accordingly. Perhaps some of our goods have been on the shelves too long. Possibly some of us are satisfied to ride complacently under our jobs instead of on top of them. Educational ideals have changed with such rapidity of late as to have become almost revolutionary. The world moves and there appears to be no such thing as fixed standards in education. Indeed anything that is static is dead or headed that way. Some of the things we still cherish are hold-overs from the day of Law and Liautard. We must ever be alert to the newer demands of a more exacting clientele and to changing standards imposed by the related sciences. Someone has said that "whatever tends to establish fixed and rigid modes of thought, tends to fossilize society." The same applies in a special way to a learned profession.

The greatest problem confronting us just now is the dearth of students in our veterinary colleges. Here we are confronted directly with the problem of remuneration. This is in line with the ever popular commencement day address, "An education for a life or a living." Leaving aside for the nonce the beauty and charm of an ideal life, with our heads in the clouds, the fact

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remains that we must eat, and remuneration concerns us greatly. Many of our young graduates being inexperienced, and financially unable to enter practice, prefer a salaried position. Indeed a considerable proportion of them, while well trained and capable, are immature and, perforce, some of them better adapted to work under direction than for themselves.

The ridiculously meager salaries paid by the U. S. Department of Agriculture is in a large measure responsible for the small number of students in our veterinary colleges. The mail carriers in my town are better paid than the men we have been graduating from a four-year professional course. We have about 200,000,000 animals in this country, valued approximately at ten billions of dollars, and less than 600 students in our veterinary colleges. There are quite as many animals now as in 1914 and only about one-fourth as many embryonic veterinarians. Perhaps the most disconcerting thing about it all is that the live stock interests manifest very little concern, insofar as we are concerned, about the future security of their investments. Somehow things just naturally refuse to be mismanaged for long and nothing arbitrary, nothing artificial, and nothing radically unjust can long endure. The recent adjustment of the salary schedule for Bureau of Animal Industry workers should have a salutary effect on college enrollment.

UNPRECEDENTED DEMAND FOR VETERINARIANS

There has been, this last year, an unprecedented demand for veterinarians to fill positions in education and research. These positions are necessarily limited. The salaries paid are fairly adequate. It is to be regretted that, because of a scarcity of trained men, it has been necessary in some instances to employ young men, recent graduates, of questionable qualifications.

Everywhere the practitioner is looked upon as representing our field in the largest scope of its possibilities. Our reputation as a profession depends in a very large measure indeed upon the practitioners and their ability to serve the public. Mark Twain facetiously remarked: "We can't all make a living taking in each other's washing, someone must saw the wood." I think we will all agree that the practitioners are our chief sawers of wood.

We all know that practitioners generally are not receiving a remuneration for their services which is commensurate with their professional attainments. Indeed an adequate remuneration, founded upon a just professional recognition, concerns us greatly.

There is no effect without a compensating cause. The rank and file of practitioners are having difficulty in selling their services for a living wage. This is more especially noticeable with a general practice in the rural districts. The laborer is worthy of his hire, but on the other hand we are confronted with the truism—"As we give so shall we receive." The live stock industry was not created for the benefit of the veterinary profession. Men are known by their works and our profession, like any other enterprise, will be fully and appropriately advertised by its achievements. Every worthwhile service has its compensation. True, "there is no use to grind unless we advertise our grist," but much depends on how we advertise. Nothing worthwhile has ever been accomplished by shouting the merits of our wares from the housetops. The goods must be delivered or they will not be paid for. A learned profession has certain standards in ethics and dignity which should be maintained. We want laws to protect us from others but not from ourselves. Legislation, resolution and printers' ink will not compel or even induce the farmer to employ a veterinarian unless he sees a financial advantage in so doing. He will not take a chance of paying a fifteen-dollar fee to save a twenty-five dollar horse. Publicity he must have, and nothing will materially alter the advertising worth of his services. Veterinary practice rests upon an economic foundation but nevertheless it is primarily altruistic and not egoistic.

SENTIMENT PLAYS IMPORTANT ROLE

Small-animal practice is succeeding best because it is to a greater degree founded in sentiment. Our sister profession will always have the advantage of us in this respect. A derelict whose soul is blackened in crime and sure to disgrace the heavenly throng enjoys the best professional and hospital attention before his uneventful departure. Our animal friends in the wide open spaces of this country mostly die without professional attention or perchance are hurried on their way from ignorant drugging with nostrums by enterprising but ignorant neighbors.

To those of us who are directing the training of young men for the veterinary profession often arises the question of preparedness. Are we meeting the needs of a clientele that is always better informed and more exacting. The curricula of our several veterinary colleges are comparatively uniform and have not been materially altered in several years. A six-year course is advocated by some, presumably the first two years to be basic, the

last four strictly professional. An optional six-year course, leading to degrees in animal husbandry and veterinary medicine, has been offered by a few of the schools but appears not to be very popular. Too many young men enter college with the primary object of obtaining a permit to try their hand, and preparedness does not then concern them greatly. It seems obvious that our problem is first to fill our veterinary colleges before seriously considering a required six-year course.

The suggestion has been made that possibly our course of instruction is too strictly professional, that training young men for citizenship is more important than training them in a specialized way for the veterinary profession. It is true, of course, that polish and refinement are indispensable assets in professional salesmanship. Education for the sake of culture alone was long the object of the academicians and anything contributing to profit was beneath their dignity. In other words culture meant a deification of those things which could not be used directly for gain. Possibly the pendulum has now swung too far the other way. Sound professional knowledge, and the scientific and biological basis for the same, is of prime importance, but an ability to meet the world on its best social plane, to solve the human equation, to cash in on the educational investment is not to be ignored in this utilitarian age. A modern conception of education recognizes the cultural value of the useful. An epicurean taste and a plebeian income is nothing short of social tragedy.

UNABLE TO SELL THEIR SERVICES

Emanuel Kant said that "education is the process whereby a man becomes a man." Where many of our practitioners are falling down is not so much from inability to diagnose and treat diseases as it is in business acumen in selling their services. The banker, the merchant, people generally, are as much concerned about his social standing, his rating as a business man and citizen as they are about his professional attainments. In these respects we can learn much from our sister profession. Someone has summarized the whole matter as "business tact and social contact." The independence of yesteryear has become the interdependence of this year. No one is sufficient unto himself alone and this is true in a special way to the one who depends upon public patronage for a living. We can make more friends by being interested in people than we can by trying to get them to become interested

in us. I feel that in a general way we are holding ourselves too much aloof from the live stock interests. Veterinarians should individually and collectively take a more active interest in breeders' and dairymen's associations and should be one of all live stock interests. In the training of veterinarians these things have their place.

The veterinarian is first of all a business man and yet we offer no business courses. The college veterinary medical association offers about the only opportunity for training in debate, essay writing, and public speaking. Nutritional diseases should receive more attention. The time devoted to anatomy, more especially the anatomy of the horse, might be reduced. It might be well made more comparative, including several species of animals to which we must now give more consideration. More time in the curriculum is needed for the study of fur-producing animals and the goat. The raising of these animals represents an industry that is now in its infancy.

A FEDERAL LICENSE TO PRACTICE

In the veterinary colleges of Europe is a vogue that possibly might well have been retained in this country. The candidate for graduation is required to take final examinations on all major subjects offered in the curriculum. Furthermore, in the British Isles the finals for admission to the Royal College of Veterinary Surgeons are conducted by the Royal Examining Board. It is very apparent that under this system the interest and efforts of students, instead of waning almost to the point of indifference in the closing months of the senior year, would receive a stimulus that would be very much worthwhile from the standpoint of preparedness. It has been tersely stated that "things that have been believed for a long time are probably not true." Conversely some things that have been discarded are again being considered in the light of their real merit. Dr. A. Liautard long advocated the creation of a national board of examiners, and no one allowed to practice veterinary medicine in this country unless he was the possessor of a degree granted by this board. This would at least solve the problem of reciprocity between the states, and do away with state examining boards. This is in substance the English system and many things can be said in its favor.

Because of the small enrollment in the veterinary courses, there is a tendency to make them more attractive by making them less

difficult—a refuge for lame ducks. Weak students drift into the easier courses.

It is true of course, in the abstract, that it is not possible for one person to educate another, and so the best we can hope to do is to offer such courses, and maintain such an educational environment as will to the highest degree encourage students to make the most of their opportunities. Teachers who can inspire students to this end are born, not made.

The profession of veterinary medicine is the youngest of the so-called learned professions. Indeed since the days of our swaddling clothes is scarcely more than fifty years. We are orienting. Youth is a period of prejudice, of bigotry, of great self-assurance, of carelessness of detail, but now as our profession has become of age, we are taking on a dignity commensurate with that of a learned profession. To keep pace with the times, standards must be raised consistently. No doubt the leaders of tomorrow in our profession will be university men trained in veterinary medicine, sanitary science, animal husbandry and agriculture.

A satisfactory and just recognition is slowly but surely dispersing the fog of ignorance and suspicion. We must prove our wares, pay the price of success in professional highmindedness and good citizenship, then appreciation must follow as the day the night. Complaining because of a lack of appreciation is cowardly and debasing; it will get us nowhere and we should have no more of it.

There is an ancient legend which seems apropos in this connection: "You will find good meat, bread and wine at Trocate, providing you take them with you." This is to teach us that we will find goodwill, honesty and appreciation wherever we go, providing we take these things with us.

DISCUSSION

DR. WILLIAM H. FELDMAN: Your Program Committee has accorded me a most unusual privilege in permitting me the honor of discussing these splendid papers on veterinary education. Frankness prompts me to say, however, that I hardly feel capable of adding anything of value to the pertinent remarks of Doctors White and Glover, who have so admirably presented their impressions after having devoted a lifetime of service to veterinary education.

However, through the kindness and cooperation of the leading veterinary educators and a few practitioners, I have collected some data pertaining to veterinary education which might appropriately be offered for your consideration at this time.

In order to convey to you the opinions given in the exact language of the writers, I trust you will bear with me while I read expressions from several of the deans of our veterinary schools. One wrote: "Too much attention is

given to graduating men who immediately after graduation become practising veterinarians." This man felt that more emphasis should be placed upon the fundamental subjects, such as pathology, bacteriology, zoology and parasitology. He closed by saying: "Elective courses for juniors and seniors should be given." Another wrote: "Taken as a whole, our veterinary curricula have already too many hours. It is exceedingly questionable in my mind whether the work required of veterinary students is of a college or university grade, when they are able to carry as many as a total of more than 160 semester credits, though the standard arts curriculum in most institutions is of only 120 semester credits duration."

One common criticism of the course of study which was made by a great many was the time allotted to anatomy at present. The majority felt that a one-year course could be arranged, that would meet the requirements. Personally, I feel that two years devoted to the study of this subject is too long, although I must admit that very few, indeed, are the graduates who possess any considerable amount of detailed anatomical knowledge. Would they know less if the course of study could be rearranged to harmonize better with our changing pedagogy? At least, the subject seems worthy of serious consideration.

The dean of one of the largest schools feels that our entrance requirements are too low, and as a consequence we attract young men of a lower intellectual capacity than the other professions. While it must be admitted that the good fellowship and square dealing accorded the student in most of our veterinary colleges often appeals to many disgruntled students, who have too often flunked out in other departments, I seriously doubt if the rank and file of our veterinary students possess a lower mentality than the average for the rest of the college or university. In support of the argument that men of unusual capacity are studying veterinary medicine are the unusual records made by two senior students in two of our schools during the past college year. One of these men received the scholarship honors out of a senior class of nearly two hundred, while the other was also the honor man of an entire institution totaling several thousand students. Yes, we have "lame ducks" in the veterinary schools but the same species can be found in the medical, dental and law schools.

Some criticism was directed, peculiar as it may seem, toward the teaching personnel of our schools. It is said that many of the teachers are of mediocre calibre, usually inadequately prepared and not the type to inspire the confidence and respect of the student. Here again, generalizations are most unjust, since all of us have known the joy of sitting under truly inspirational personalities, whose command of the subject matter could not be questioned. It is true that some are teaching who, by temperament, have no place in the lecture-room, and there are many others who are woefully deficient in the knowledge of their subjects, but the bulk of the fault lies in this instance, not with the administrators of the veterinary schools, but with the niggardly attitude often imposed upon them by the so-called economy programs of those who control the purse-strings of the college or university of which the veterinary school may be a part. There is no question but that the faculties of our veterinary schools are for the most part shamefully underpaid.

Several expressed the opinion that too much time is being consumed with subjects that should be considered prerequisite to the study of veterinary medicine. As the dean of one of our oldest schools put it: "I feel that students who are to enter veterinary medicine should have a thorough training, not only in English and possibly foreign languages, but especially in the sciences of chemistry, botany, zoology, and the like. These I do not feel have a rightful place in the professional curriculum." This brings one to a phase of veterinary education which is perhaps the next step. I mean the extending of the course of study from four to five or six years. Some schools are offering five- and six-year courses at the present time, and while these courses have attracted only a few to date, I feel that if the future veterinarian is to assume his rightful place with the other learned professions, and keep abreast of the related sciences he must prepare himself in a manner comparable to those who enter the medical profession. Practically, of course, it is not feasible under the present conditions to insist on an extension of the time necessary to qualify as a graduate veter-

inarian, but all will agree that stricter standards and more education have given us better physicians and by the same token the character of veterinary service will be correspondingly elevated when our graduates have the unquestionable benefits of more and better training.

In this connection the propriety of conferring a doctorate degree for four years of college work opens a serious question, and one which the Committee on Education should consider in its future recommendations. It would seem more in keeping with present academic practice to offer a Bachelor of Science in Veterinary Medicine to those who complete the four years' work, the Master's degree to be given to those who elect to complete five years, and, to those who do graduate work two years in excess of the first four, a doctorate degree of real significance could be properly conferred.

That the methods of instruction in vogue at the present time are not above criticism was reflected in a most interesting communication received from a man who made a splendid scholastic record while at college and who was that type of student whose mind turns to the practical. He made a list of the things he had never done in college. These included:

1. Never passed a stomach-tube.
2. Never made an intradermic tuberculin test.
3. Never made an intestinal and gastric lavage in the dog.
4. Never done any meat inspection.
5. Never caponized, or saw it done.
6. Never done any intravenous medication.

He closed by saying: "I could name a long list, but the above is sufficient to show that the new graduate is not prepared to make a practical application of the theories he was taught." This is a serious indictment, and it behooves our veterinary schools to take an inventory as Dr. Glover suggests and see if adjustments cannot be made which will better train a higher type of man to discharge creditably the increasing responsibilities demanded of every member of the profession.

In answer to the question as to whether the curriculum should be standardized, one of our foremost educators recommended that this committee "prepare a plan of instruction thorough enough so that if carried out, the D. V. M. degree would be recognized by all members of the American Association of Universities as fulfilling all the requirements for admission to graduate work and the full candidacy for the Ph. D. degree."

This is a suggestion of the first importance, especially to our graduates who desire to pursue graduate study for advanced degrees in institutions other than the one from which they obtained their veterinary degree.

Many other worthy suggestions were made by men high in our educational field, but since I have already taken more than the allotted time, I feel that I should close. There are many others here who would no doubt like to discuss this subject.

Dog Trials to End in Kentucky

It is reported that Kentucky's trials for dogs, which have established court history and caused comment the country over, are destined to end if a ruling handed down in Fayette Circuit Court recently by Judge J. Keene Daingerfield is upheld in the Court of Appeals.

Judge Daingerfield held that the old law which provided for "murder trails" of dogs under sheep-killing charges has been repealed by an act of 1918 relating to the licensing of dogs. The Blue Grass State has seen several historic dog trials.

THE TRANSMISSIBILITY, INFECTIVITY AND SENSITIVITY OF TUBERCULOSIS*

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INTRODUCTION

Consumption and phthisis of the human race have existed since the dawn of civilization. Pearl disease of ruminants has been recognized for centuries, but spotted livers and nodular intestines of fowls are conditions of comparatively recent recognition. It is now definitely established that all of these disease conditions are manifestations of the pathogenic activities of the various types of *Mycobacterium tuberculosis*, resulting in that specific microbial disease, tuberculosis.

If we add to this significant fact the transmissibility of the disease to animals experimentally, in 1865; the discovery of the tuberculosis organism and its propagation by artificial means, in 1882; and the preparation of the first tuberculin as a diagnostic agent, in 1890, we have enumerated the foremost and outstanding milestones along the pathway of tuberculosis history down to the present generation.

However enlightening and significant these milestones are, the year 1917 initiated a period in tuberculosis history that is destined to mark an epoch of far-reaching importance in the annals of this disease. It will be remembered that the Accredited Herd Plan had its birth at that time and was the forerunner of the Area Eradication Plan and other related projects, which go to make up the nation-wide tuberculosis eradication campaign, the most magnitudinous sanitary project of all time.

A program of such magnitude is not without its puzzling perplexities, grave responsibilities, and almost unsurmountable difficulties. The sanitarians of the country were confronted with a live stock population deeply rooted, from many years' implantation, with an infection which will require tremendous sums of money and years of patient waiting on the part of the public before it can be carried through to satisfactory completion.

While each milestone of progress, previously referred to, may be considered an epoch in itself in tuberculosis history, which in turn gave its material contribution of basic knowledge to the

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problem complex, the present period found many things of a very fundamental nature calling for solution before such a campaign could be carried to a conservative and efficient conclusion.

However, we are approaching the problem in a diligent and enthusiastic manner. The very status of affairs and the magnitude of the work seem to have given unusual impetus to all concerned in the great tuberculosis movement, which has never had a parallel in sanitary science. Reluctancy and hesitancy are manifested only by a minor few, who have failed to become imbued with the spirit and importance of the movement, and the work is going forth with an undeniable force and firmness which is sure to terminate in success.

An overwhelming majority of the live stock fraternity are according whole-hearted support and co-operation in the campaign. The federal and state sanitarians and field veterinarians in general are applying themselves with a determination to "complete the job," and laboratory workers on many sides are availing themselves of all possible opportunities to make further researches into unknown fields of this disease. In fact, some investigators have temporarily suspended projects that they have been pursuing, that they may give special studies to this timely subject.

OLD THEORY UPSET

The nation-wide tuberculosis campaign was designed and initiated primarily for the purpose of eradicating bovine tuberculosis from our cattle and swine herds. The program had hardly reached the initial stages of progress when it was learned that complications of a grave nature were involved. The old theory and teaching "that the eradication of tuberculosis from cattle would automatically eliminate the disease from swine" was no longer tenable and many of those concerned about the laboratory phases of the problem began to wonder about the possibilities of the transmissibility of the various bacillary types of the tuberculosis organism among our domestic live stock, including fowl and possibly the human family.

This phase of the problem called for more enlightenment before the most intelligent measures could be formulated and the most efficient sanitary policies put in force. The bacillary types which are definitely known to produce the classical disease in man, beast and fowl are those of human, bovine and avian strains. If these types would confine their pathogenic and antigenic proclivities to their natural or optimum hosts, the problem of eradication

would be greatly simplified. However, the possibility of them invading the fields of foreign host species, more extensively than had previously been anticipated, now became more probable, and to that end some very worthy projects have been in progress during the last decade.

TRANSMISSIBILITY STUDIES FRUITFUL

Before the subject of intertransmissibility can be penetrated very deeply, it becomes quite essential that one knows almost absolutely the bacillary strains with which he is dealing. While microscopic examination of stained preparations and careful studies of the morphological characteristics of the various types by cultural methods afford some means of differentiation, it remains for the experiment-animal-inoculation method—cavia, rabbits and the common barnyard fowl—for the most reliable and dependable way of distinguishing bacillary strains. This method however cannot be expected to ferret out accurately all the so-called intermediate, transitional or aberrant types of the organism. Thus, by carefully planned exposure trials, by actual ingestion of tuberculous materials and by direct inoculation of tuberculosis viruses, together with accurate typing of the bacillary strains of the organism involved, we are able to arrive at a fairly satisfactory conception of the transmissibility and intertransmissibility of the various types of the organism among man, beast and fowl.

Foremost perhaps, at least from a biologic, economic and sociologic standpoint, is the question of transmission of the bovine and avian infections to the human family. Although man has not been exposed purposely, made to ingest forcibly, or inoculated premeditatively with these specific types of tuberculosis, nevertheless, he has unknowingly served as an experiment animal par excellence, for this special purpose. Countless peoples throughout the country are consuming raw, unpasteurized milk from untested cattle and meat products emanating from establishments that do not maintain veterinary inspection. Thus, the opportunities for obtaining the germs from these sources are ideal in many respects.

While these conditions have prevailed for centuries, scientific inquiry has been made into the problem only during the last few years. These investigations have revealed the facts of the case and unimpeachable evidence is now at hand from the accumulated data which very conclusively proves that bovine tuberculosis is

transmissible to the human race in no small measure. Innumerable statistics could be cited to substantiate this contention. However, a few gathered at random will suffice to illustrate the point in question.

CLASSICAL STUDIES OF PARK AND OTHERS

Figures gathered from a large number of hospitals throughout the land from children under five years of age, where cows' milk constitutes the major portion of the diet, showed that from 4 to 70 per cent of the cases of tuberculosis were of bovine origin. Dr. W. H. Park, of the New York City Board of Health, estimates that 300 children die annually from bovine tuberculosis in that city. Dr. A. Stanley Griffith, of the Royal British Tuberculosis Commission, found 221 (20.7%) of 1068 cases of human tuberculosis of bovine origin. Of these 221 cases, 83 (37.55%) were of children under five years of age. He reports further, of 312 children between five and ten years of age, that 92 (29.45%) manifested the bovine type of germ. Dr. M. J. Rosenau, of Harvard Medical School, finds in assembling the data from several investigators that 408 (16.2%) of 2527 cases studied proved to be of bovine infection and Chesley, of Minnesota, finds that in children 23.8 per cent of generalized tuberculosis, 40 per cent of tuberculosis of cervical glands and 49 per cent of abdominal organs are the results of infection with the bovine organism. These figures are convincing and need no further comment or explanation for those of average intelligence. They may, however, require special interpretation to the various anti-propagandists, the self-styled broad, comprehensive-minded humanitarians who piddle away their time and lives cluttering up progress in public health and live stock sanitation work.

Aside from the possibilities of the bovine type of organism in the juvenile period of human life, it accounts for practically all tuberculosis infection in its natural host species, the bovines; the calf being unusually susceptible to its ravages. Further, it is responsible for the major portion of infections in the hog, horse, goat, cat and possibly the sheep. However, the latter species requires further investigation, as quite recently the Nebraska Station found one case in sheep which proved to be of human origin while the North Dakota Station typed two cases which were caused by the avian type. A limited number of cases in the dog reveal the fact that both the bovine and human types are about equally involved in that animal. It is the opinion of the

writer that considerable more typing work should be done in cases of tuberculosis in the horse, sheep, goat, dog and possibly the cat. We can then speak, perhaps, with more assurance and definiteness as to the kind of infection in these classes of animals. According to our present status of knowledge, the bovine strain of tuberculosis has a much wider distribution in the animal world than either the human or avian types of the disease.

ACTIVITIES OF THE HUMAN TYPE

In summing up the activities of the human type of organism, it is readily observed that its pathogenic proclivities are limited to more narrow confines than those of either the bovine or avian strains. Of course, as we may expect, it is the principal cause of the classical disease in the human family. It likewise accounts for practically all tuberculosis infection in the various species of monkeys that have been investigated. The available evidence indicates that this type is non-pathogenic for bovines, as numerous studies conducted on this phase of the problem show that they are quite resistant to that organism. It would appear as though swine are relatively immune to the human germs.

Outside of an outbreak of tuberculosis in swine, feeding on garbage from a tuberculosis sanitarium, investigated by the Montana Livestock Sanitary Board, the human bacillus has not been demonstrated in this class of live stock from the many cases of tuberculosis that have been carefully typed throughout the country during the last few years.

The avian bacillus is the youngest or latest branch to spring from the tuberculosis tree. This type of the disease has been recognized for only about three-quarters of a century. It has been given but meager experimental consideration until the last few years, when the subject has taken on a tremendous stimulus and interest and is now attracting unusual consideration on the part of research workers in tuberculosis. It plays such an important role in the program of the universal tuberculosis eradication campaign, now in progress, that it is certain to command a very prominent and essential place in the present era in tuberculosis history.

As we would naturally presume, the avian organism manifests its greatest and most consistent activities in the members of the avian family. It more particularly confines its depredations to the gallinaceous birds, making its greatest onslaughts on the domestic fowl of this class and those of the field and forest when

confined in captivity. The disease is extremely rare in aquatic birds, even when subjected to strict confinement for long periods of time. The disease is becoming exceedingly commonplace among chickens, turkeys and guinea fowl in many sections of our country.

Next in order of importance to fowl infection, and possibly parallel to it from an economic view point, is the invasion of swine by the avian disease. Carefully controlled experiments reveal the fact that swine, particularly young pigs, are extremely susceptible to the avian bacillus. The latter will become infected quite readily upon slight provocation, when exposed to the droppings, the diseased organs and infected carcasses of tuberculous fowls. We further gather from our experimental tuberculosis barnyard studies that pigs as well as chickens will contract the disease in the barnyard, during spring and early summer, which had harbored tuberculous birds the previous summer and fall. It is our opinion that the young pig will serve equally well as an experiment animal for avian tuberculosis as the natural host species, the common barnyard fowl itself, as regards localized infection. Typing experiments conducted at the Nebraska, Illinois and North Dakota Experiment Stations showed that from 60 to 88.5 per cent of all cases of localized tuberculosis of swine examined were of avian origin.

THE STATUS OF AVIAN INFECTION IN CATTLE

Regardless of the many speculations made and the adventurous theories advanced regarding avian infection in cattle, carefully controlled barnyard experiments fail to reveal any evidence whatever that the possible intervention of the avian bacillus in this class of live stock has attained economic proportions. Cattle can be and frequently are sensitized by the avian type of organism from cohabitation with and exposure to infected birds. From the data collected to date, it develops that infection leading to lesion disease in cattle from this strain of the organism is extremely rare.

We have repeatedly succeeded in readily sensitizing cattle with the avian virus by inoculations into the scarified skin and instillations into the conjunctival sac as well as by exposure methods. The former can be accomplished with dosages of .1, .01, .001 and .0001 milligrams of the avian virus. Such sensitizations are but temporary, however, as they practically all disappear within a few months after the inoculations or removal from the exposure.

An exceedingly encouraging feature regarding the phenomenon of sensitization of cattle with avian tuberculosis lies in the fact that they are apparently but rarely detected by the mammalian tuberculins. In our own experiments, where hundreds of tests have been made on such animals, the mammalian tuberculins invariably fail to elicit reactions, whereas the animals readily respond positively to the avian tuberculins that have been tried. This condition would appear to preclude the possibility of no-lesion cases, resulting from avian-sensitized cattle, reacting positively to mammalian tuberculins.

TYPING EXPERIMENTS BEING CONDUCTED

Our knowledge of the occurrence of avian tuberculosis in man or its possible transmission to the human race is entirely too meager. Perhaps the most noteworthy work on the subject is now in progress in the form of a series of typing experiments of surgical cases of human tuberculosis at the Nebraska Experiment Station. From their latest report of progress that has come to our attention, more than sixty cases have been typed and not a single one complies with the avian strain. While these data are comparatively few, they indicate in no uncertain terms the trend of the problem.

Pigeons and sparrows, which may be considered more or less pestiferous birds and which may infest practically every farm premise, are susceptible to avian tuberculosis. Although we failed for four years to increase the percentage of avian infection in these birds while cohabitating with tuberculous chickens in our experimental tuberculosis barnyard, we did succeed in proving conclusively that both were mechanical carriers of the disease from infected to healthy flocks.

We gather from the literature that the common rat and mouse have manifested spontaneous avian tuberculosis. During the last five years, at the North Dakota Experiment Station, we have made a rather determined effort to verify those observations, but have failed absolutely. Our greatest endeavors have been with the common rat. We have continuously harbored them with large numbers of tuberculous chickens, from birth to time of death, covering months and years. We have repeatedly inoculated them subcutaneously, intraperitoneally and intracardially with fresh lesions and various cultures and all have failed to result in infection. However, we had no difficulty, whatever, in demonstrating that the common rat is a very potent mechanical

confined in captivity. The disease is extremely rare in aquatic birds, even when subjected to strict confinement for long periods of time. The disease is becoming exceedingly commonplace among chickens, turkeys and guinea fowl in many sections of our country.

Next in order of importance to fowl infection, and possibly parallel to it from an economic view point, is the invasion of swine by the avian disease. Carefully controlled experiments reveal the fact that swine, particularly young pigs, are extremely susceptible to the avian bacillus. The latter will become infected quite readily upon slight provocation, when exposed to the droppings, the diseased organs and infected carcasses of tuberculous fowls. We further gather from our experimental tuberculosis barnyard studies that pigs as well as chickens will contract the disease in the barnyard, during spring and early summer, which had harbored tuberculous birds the previous summer and fall. It is our opinion that the young pig will serve equally well as an experiment animal for avian tuberculosis as the natural host species, the common barnyard fowl itself, as regards localized infection. Typing experiments conducted at the Nebraska, Illinois and North Dakota Experiment Stations showed that from 60 to 88.5 per cent of all cases of localized tuberculosis of swine examined were of avian origin.

THE STATUS OF AVIAN INFECTION IN CATTLE

Regardless of the many speculations made and the adventurous theories advanced regarding avian infection in cattle, carefully controlled barnyard experiments fail to reveal any evidence whatever that the possible intervention of the avian bacillus in this class of live stock has attained economic proportions. Cattle can be and frequently are sensitized by the avian type of organism from cohabitation with and exposure to infected birds. From the data collected to date, it develops that infection leading to lesion disease in cattle from this strain of the organism is extremely rare.

We have repeatedly succeeded in readily sensitizing cattle with the avian virus by inoculations into the scarified skin and instillations into the conjunctival sac as well as by exposure methods. The former can be accomplished with dosages of .1, .01, .001 and .0001 milligrams of the avian virus. Such sensitizations are but temporary, however, as they practically all disappear within a few months after the inoculations or removal from the exposure.

An exceedingly encouraging feature regarding the phenomenon of sensitization of cattle with avian tuberculosis lies in the fact that they are apparently but rarely detected by the mammalian tuberculins. In our own experiments, where hundreds of tests have been made on such animals, the mammalian tuberculins invariably fail to elicit reactions, whereas the animals readily respond positively to the avian tuberculins that have been tried. This condition would appear to preclude the possibility of no-lesion cases, resulting from avian-sensitized cattle, reacting positively to mammalian tuberculins.

TYPING EXPERIMENTS BEING CONDUCTED

Our knowledge of the occurrence of avian tuberculosis in man or its possible transmission to the human race is entirely too meager. Perhaps the most noteworthy work on the subject is now in progress in the form of a series of typing experiments of surgical cases of human tuberculosis at the Nebraska Experiment Station. From their latest report of progress that has come to our attention, more than sixty cases have been typed and not a single one complies with the avian strain. While these data are comparatively few, they indicate in no uncertain terms the trend of the problem.

Pigeons and sparrows, which may be considered more or less pestiferous birds and which may infest practically every farm premise, are susceptible to avian tuberculosis. Although we failed for four years to increase the percentage of avian infection in these birds while cohabitating with tuberculous chickens in our experimental tuberculosis barnyard, we did succeed in proving conclusively that both were mechanical carriers of the disease from infected to healthy flocks.

We gather from the literature that the common rat and mouse have manifested spontaneous avian tuberculosis. During the last five years, at the North Dakota Experiment Station, we have made a rather determined effort to verify those observations, but have failed absolutely. Our greatest endeavors have been with the common rat. We have continuously harbored them with large numbers of tuberculous chickens, from birth to time of death, covering months and years. We have repeatedly inoculated them subcutaneously, intraperitoneally and intracardially with fresh lesions and various cultures and all have failed to result in infection. However, we had no difficulty, whatever, in demonstrating that the common rat is a very potent mechanical

spreader of the disease to both chickens and swine. We further learned that the avian organisms are able to withstand the passage of the alimentary tract of the rat in a viable condition and capable of producing infection in both chickens and swine.

Among the experiment animals the behavior of the various bacillary types is quite constant, exhibiting their pathogenic propensities in a rather definite and fixed manner. Cavia and rabbits are particularly susceptible to the bovine type but fowl are practically immune to same. The human strain is unusually pathogenic for cavia but only slightly so for rabbits and inactive in fowl. Lastly, the avian type attacks fowl in the classical manner and shows marked pathogenicity for rabbits, but fails to produce infection in cavia.

SENSITIZATION AND INFECTION

Intimately associated with and practically parallel to the transmissibility phase are the factors of sensitivity and infectivity of the different host species with the various types of *Mycobacterium tuberculosis*.

The term sensitization is generally interpreted as a state of body tissues, resulting from the pathogenic and antigenic activities of various agencies, such as micro-organisms, parasites and proteins in general. In the bacterial field in particular, it is quite generally accepted that the state or condition of sensitization must be attended by either gross or histopathologic lesions. In this restricted sense sensitization must necessarily be preceded or accompanied by infection.

On the other hand we recognize true infection as a condition of body tissues caused by the multiplication and propagation of pathogenic organisms to the extent that they produce manifest lesions, either macroscopic or microscopic in size. Expressed otherwise, infection is a visible, material manifestation of the causative agent, whereas sensitization is an invisible, immaterial state that can be demonstrated only by biologic or allergic reactions. Sensitization may be and presumably is the result of true lesion infection, in a majority of bacterial diseases, but we do not believe it is necessarily so.

POSSIBLE SENSITIZATION WITHOUT LESION INFECTION

In a consideration of some of the means of sensitization in the absence of real lesion infection, we cannot overlook the so-called Yersin type of tuberculosis, occasionally encountered in fowl. These birds respond with typical positive reactions when sub-

jected to the tuberculin test. However, upon autopsy they are always characterized by the absence of specific lesions, either macroscopic or microscopic in nature. The reactions are evidently the result of sensitization caused by the metabolic products of the tuberculosis organisms, which can be demonstrated in the liver tissue of many such positive-reacting, no-lesion cases.

Further, let us cite the case of tuberculins. Practically all veterinarians who have had extensive experience with tuberculin-testing of cattle, especially with the subcutaneous method, are aware of the danger and unreliability of retests conducted too soon after a previous test has been made. Such tuberculinizations or inoculations of tuberculin often result in sensitization of the animal.

Tuberculin is an artificial, laboratory preparation. Its active constituents consist chiefly of the metabolic products of the tuberculosis organisms. In the case of lesion infection in the animal, each focus becomes a miniature tuberculin laboratory of itself. The metabolic products, containing the potent constituents, are given off from the lesions and the animal tissues become sensitized in the usual way. Thus we see that sensitization in tuberculosis can be attained in at least three ways, viz., by lesion infection, by the injection of tuberculins, and by the Yersin-type method previously described.

RELATIONSHIP OF SENSITIZATION TO THE NO-LESION CASE

First, it must be acknowledged that the so-called no-lesion case may be more theoretical than real. Personally, I do not believe it possible for anyone, under the most detailed and searching methods, to determine absolutely, in all instances, the presence or absence of minute lesions in a positive-reacting animal. However, with this thought uppermost in mind, I am of the firm belief that we have more or less specific sensitization to tuberculosis in cattle without demonstrable lesion infection.

In support of this statement, we have considerable data available concerning the avian germs and a limited amount regarding the bovine organisms in cattle.

As reported in a previous communication, we have repeatedly succeeded in sensitizing cattle with avian tuberculosis by cohabitation with tuberculous birds, by feeding the droppings and diseased organs, by rubbing the germs into the scarified skin and by instillation of the avian virus into the conjunctival sac. The latter two conditions were accomplished with dosages as low as .0001 mg. of the avian cultures.

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In practically all of the cases autopsied, no lesions could be observed. Some of the animals were kept for two and three years and resensitized a second and third time by subsequent exposure and no lesions could be demonstrated at autopsy.

These consistent findings, associated with the fact that the sensitization was in all cases of short duration and of a temporary nature, fading out entirely within a few months after being inoculated or removed from the exposure in our experimental tuberculosis barnyard, are apparently significant in themselves.

SENSITIZATION EXPERIMENTS

Last summer a similar experiment was initiated with the bovine type of organism in which twenty-five young cattle were the recipients. The inoculations were made into the abraded skin and the conjunctival sac and the dosages of virus ranged from .01 to .0001 mg. Seventeen of those animals became positive reactors to mammalian tuberculin. Of these, fourteen became lesion-infection cases, as manifested at autopsy from five to eleven months afterwards. The remaining three animals reacted positively for two to three months, then ceased to react. At autopsy no lesion infection could be demonstrated. This same experiment is in progress the present season, with an additional twenty-five young cattle. We expect to continue these investigations for some years, with the hope that considerable additional data can be obtained upon this very important phase of our problem. While these data are insufficient from which to draw definite conclusions at this time, they certainly provide potent material for most serious consideration for those engaged in unravelling some of the mysteries of the tuberculosis problem complex. What bearing, if any, do they have upon the sensitization phenomenon, the no-lesion, reacting animal and ultimately the tuberculosis problem? We appear to have rather definite proof that sensitization can be attained by the introduction of tuberculin into the animal body in which we are positive that lesions play no active part. Secondly, we further know that sensitization is acquired in fowls by the Yersin type of the avian organism which fails to colonize and produce lesion infection.

We take it for granted that some animals on infected farms with contaminated environments have ample opportunities to receive into their bodies, in some manner, the small loads of tuberculosis virus referred to above, either via the conjunctival sacs, the abraded or broken skin and more probably the alimentary tract.

Is it not possible and quite probable that these organisms in their stay or passage through these ports of entry are giving off their metabolic products in sufficient quantities to sensitize the tissues adequately to elicit positive reactions with homologous tuberculins, without establishing true lesions? If this condition obtains, which does not seem impossible from the available data we have before us, it appears as though it would explain some of our no-lesion cases, to say the least.

LIMITED KNOWLEDGE OF VIRULENCE

The brief discussion which has been given on the transmissibility, infectivity and sensitivity of tuberculosis is but a mere item as compared to the whole story. It can be readily observed that the degree and extent to which these phenomena assert themselves in a given host species, are largely determined by the state and condition of the organism itself. The deeper we delve into the tuberculosis problem the more fully we appreciate the incompleteness and meagerness of real knowledge as pertains to the more detailed characteristics of the tuberculosis organisms. Our present conception of virulence is entirely too limited. Our measure of virulence is but arbitrary. Both the biological and chemical aspects of the organism require more searching investigation before we can hope to establish a more definite or standard measure of virulence. It is not only highly desirable but almost absolutely necessary to have more accurate means of estimating, grading, weighing or measuring the virulence of the *Mycobacterium tuberculosis*. With this very important factor more definitely established, I firmly believe that much greater progress will be made in solving some of the apparent idiosyncrasies shown by some of the so-called intermediate, mixed, or aberrant types with which we now have to contend.

DISCUSSION

DR. L. ENOS DAY: In the experiment with the twenty-five cows, I would like to know whether you are using the avian or bovine type of organism.

DR. SCHALK: The bovine type.

DR. DAY: You mentioned something about weight.

DR. SCHALK: Those weights or dosages were calculated from cultures on solid media.

DR. E. A. WATSON: Dr. Schalk mentioned the subject of temporary sensitization. I would like to ask, when sensitization has ceased after a tuberculin reaction, whether he considers the animal is free from infection. We have often been misled by being in too much of a hurry to kill our animals to find out whether they have tuberculous disease or not. We have killed guinea pigs in six or eight weeks after inoculation and in the absence of lesions and of tuberculosis we are apt to say it is not infected. A guinea pig may live

a year or even longer and then come down with generalized tuberculosis. In other animals, besides guinea pigs, we must take into consideration the factor of time, and it is a very important one.

Another question on sensitization. Sensitization, as we all admit, is dependent upon infection. Can you have infection without sensitization? We perhaps, emphasize too much "no-visible-lesion" cases and worry too much about them. Why should we expect to find lesions in all cases of infection? We would be led to believe that a tuberculin reaction is a reaction to tuberculous disease. The more serious thing is failure to react when infected. European authorities claim there may be a form of infection, a purely parasitic type, without lesions and consequently without the tuberculin reaction. In these so-called breaks in accredited herds, it is not always the fault of the owner of the herd, because he has broken the regulations and fed milk that has not been pasteurized. I think it is possible that there are cases of infection which do not react to tuberculin tests, and that there is a difference between sensitiveness and relative immunity or resistance as indicated by the tuberculin test.

We have done a little work in avian tuberculosis and made a very interesting finding this spring. On the shores of Lake Erie, when the birds migrate up there in the springtime, there are a number that die, and we occasionally have birds sent to us for examination. This spring one of the naturalists in that part of the country sent us a number of dead crows. He wanted to know why they were dying. He said they had the appearance of chicken roup. It is true they had. But in addition, we have found already, in about fifteen crows, typical tuberculosis. We have caught crows around the Research Station. We managed to bring down twenty. In these Dr. Mitchell found three cases of tuberculosis. It is a very interesting fact. We are trying to type the bacillus found in each of the crows. One appears to be the bovine type. Another produces generalized tuberculosis in poultry and guinea pigs. When Dr. Schalk spoke of rats in connection with infection, it occurred to that the crow might be involved in the spread of bovine and swine tuberculosis.

DR. SCHALK: Dr. Watson has brought out, what I consider, some intensely interesting phases bearing upon the tuberculosis problem. The first question—temporary sensitization. We have done but very little work with the bovine type of the organism. We had a herd of twenty-five young cattle last year and a similar number the present year under experimentation. The results of those investigations are incomplete and not ready to be given out at this time. Suffice it to say that one animal (yearling calf) during the present season has become sensitized from one-one hundred thousandth part of a milligram of bovine tuberculosis virus as evidenced by positive tuberculin reactions. Beyond this the foregoing remarks that I have made pertain entirely to sensitization of young cattle with the avian germs.

We use the term temporary sensitization because that is exactly what the condition of these sensitized cattle appears to be. It was arrived at and used only after considerable experimentation. For instance, approximately 80% of yearling calves cohabiting with tuberculous fowl become sensitized with avian tuberculosis or rather develop avian tuberculin allergy or they react positively to avian tuberculin. This sensitization or allergy to avian tuberculin disappears or fades out within a few months after the animals are removed from the avian tuberculosis exposure. These same animals as two-year-olds were exposed a second time and again became sensitized or reacted positively to avian tuberculin. When again removed from the avian tuberculosis exposure the sensitization disappeared a second time within four months. Lastly, these same animals as three-year-olds, which had been previously sensitized twice, were subjected to a third exposure to tuberculous fowls and again became positive reactors with avian tuberculin. At the close of this 3-year experiment the animals were slaughtered and most searching autopsies held and no evidence whatever of lesion infection observed. It will be observed that some of these animals became sensitized as yearlings and two-year-olds, thereby providing sufficient time for lesion development.

DR. WATSON: Were the sensitizing bacilli still present?

DR. SCHALK: We do not definitely know. During the earlier years of our avian tuberculosis studies in cattle in which an overwhelming majority of the sensitized animals exhibited no evidence of lesion infection we put forth every effort to locate minute lesions or show the presence of the avian tuberculosis organisms. Experimental animal inoculations, cavia, rabbits and common barnyard fowl, with various lymphoid tissues of the sensitized animals failed to elicit the presence of viable organisms. In our later experiments we did not resort to experimental animal inoculations with the apparent negative tissues. We did not think from our earlier findings that it was justified. The procedure simulates so much, the old proverb "looking for a needle in a haystack."

DR. WATSON: When do you consider you have infection without sensitization?

DR. SCHALK: I do not want to be misconstrued in this matter. I am not making a definite statement that we do have sensitization without infection. I think we are all quite clear as to the orthodox teachings on the subject of sensitization and infection; i. e., that all sensitization is preceded by or accompanies infection. However, does that make it absolutely true?

Knowledge, especially scientific knowledge, however well established and universally acknowledged, in most instances at least, I believe, should be considered as relative and not absolute. Present-day knowledge is simply the known facts in the case as established up to this time. Knowledge is usually a growing, a progressive commodity to which contributions are made from time to time. Therefore, is it not possible that some future generation will explain in some manner how organisms in their passage through the animal body may and do sensitize their hosts without establishing manifest lesion infection? Although such organisms may not propagate sufficiently to produce local lesions, their physiologic, metabolic functions will remain more or less active throughout their life or their stay in the body and in this manner may sensitize their hosts. And so, may it not be with avian tuberculosis organisms in cattle?

We do have an abundance of evidence at hand showing that cattle cohabiting with tuberculous fowls become sensitized to avian tuberculosis. This allergic state presumably largely obtains from the animals ingesting the organisms in the droppings of the infected birds. The foremost question in our minds is, just how do these animals become sensitized? Of course some are prone to dismiss the question by stating that there are minute, invisible lesions present that are not observed. That might possibly be, but when one consistently encounters seemingly lesionless cases in numbers of cattle year after year and finds the sensitization of a more or less temporary nature, I believe that one is highly justified in raising the question of possible sensitization without lesion infection.

DR. ROBERT GRAHAM: How many times, Dr. Watson, have transfers been made from crows where lesions were found?

DR. WATSON: We are carrying the strains in guinea pigs and rabbits. The bovine strain has had five or six passages. The other strain is carried in guinea pigs and chickens and produces generalized tuberculosis.

DR. DAY: I was very much interested in what Dr. Watson said concerning latent tuberculosis. I believe tubercle bacilli may at times remain in the bodies of animals for a long time before they become sensitized and react to tuberculin.

Monkey Loses a Home

A Detroit woman going south wanted to give her pet monkey to the Detroit Zoological Park. When Director John T. Millen said he could not promise a daily "hot water and soap bath," the monkey-owner indignantly hung up the telephone with the remark, "Cleanliness is next to Godliness, even for a monkey."

THE PATHOLOGY OF SWEET CLOVER DISEASE IN CATTLE*

By LEE M. RODERICK, *Fargo, N. Dak.*

North Dakota Agricultural Experiment Station

It was definitely determined some years ago that damaged sweet clover in the form of either silage or hay was responsible for the deaths of cattle from fatal hemorrhage. It was at once apparent that a new disease was involved. The history, occurrence and pathology of the condition were so striking that a great deal of interest was aroused. Moreover, these losses had become numerically greater with the increasing utilization of sweet clover as a forage crop.

Research work was started on the problem at the North Dakota Agricultural Experiment Station during the winter of 1921-22. Credit is due to our Canadian co-workers, however, for first attributing those mysterious losses to spoiled sweet clover hay. It is now intended to present some of the details of that study, which has been one of our experimental projects since its inception. I do not propose to give a general discussion of the disease nor to cover the entire field of the investigation. It will be limited, therefore, to some features of the effect of the damaged sweet clover on the animal mechanism.

ETIOLOGY

Obviously, one of the first questions asked concerning a new disease is with reference to its etiology. The causal role of the sweet clover is unquestioned. It is not a transmissible infectious disease in the usually accepted sense. Further work is required, however, to explain satisfactorily the alterations which take place in the sweet clover and thereby cause the disease. There seems to be no available information to indicate that this characteristic fatal hemorrhage can arise from other forages. It seems to be definitely limited to sweet clover and only when in the form of spoiled hay or silage. No attempt will be made to discuss symptomatology and clinical features of the disease in this paper in any detail.

HEMORRHAGE

Hemorrhage is the characteristic lesion of the disease which results from the feeding of damaged sweet clover hay or silage.

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The progressive delay in the coagulability of the blood of the animal which is fed on such forage is the first recognizable functional alteration and is always present as shown by experimental work. The hematology and physiologic factors involved in the altered coagulation process are entitled to further and more detailed consideration at another time. Death may occur from surgical wounds externally when the blood is in the imperfectly coagulable state or from hemorrhage into the tissues.

Hemorrhage has been found in practically all parts of the body. It has been seen in the lateral ventricle of the brain, in the wall of the bladder, and in the medullary cavity of the long bones, but

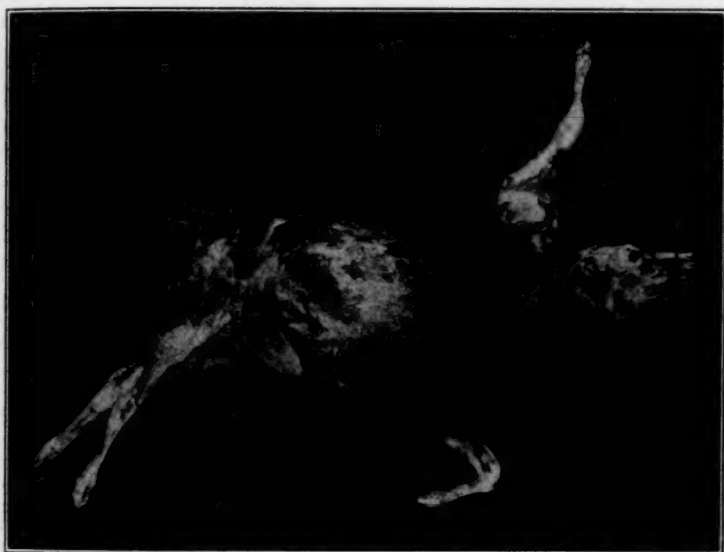


FIG. 1. Fatal hemorrhage from sweet clover.

the subcutaneous and intermuscular fascia is a far more common location. Common sites of hemorrhage are the ischial and thigh regions, the neck and shoulders and the wall of the thorax. It may assume the form of a massive hematoma, which may contain twelve quarts of semi-fluid blood or be interposed as sheets between the muscles. A layer, two to three inches in thickness, may be found beneath the scapula or between the abdominal muscles. It has occurred in the broad ligaments of the uterus of a parturient cow. Hemorrhage is common on serous surfaces and more rare in the mucosa although epistaxis and bloody milk are seen.

Hemorrhage in the parenchymatous organs is rare. The writer has never seen it in the kidney but it is not uncommon in the perirenal fat. The lateral and ventral borders of some of the intermuscular and subcutaneous hemorrhages are often bordered by a yellow margin. Schofield,¹ who published the first technical paper on this problem, mentions edematous swellings. The writer has never seen an edema of the limbs or one which did not include a contiguous hemorrhage. It is probable that such areas involve a spread of a gradually increasing perme-



FIG. 2. Rumen with characteristic hemorrhage.

ability of an area of the vascular bed, wherein the red cells are retained at the border or it is partly the gravitation of the serum from the mass of the hemorrhage.

Death seems to be due only to the loss of blood and especially of the red cells from the circulatory system. The clinical symptoms substantiate that assumption. A Colorado cattleman dehorned 80 calves fed on sweet clover and 65 died of hemorrhage within a day or two. An apparently healthy bovine may develop clinical symptoms of hemorrhage into the tissues and die within a few hours. The active, well-developed calf born of a cow fed

for thirteen days on damaged sweet clover hay dies of hemorrhage twenty-eight hours after birth. Twenty-five young bulls, varying in age from eight to fourteen months, were castrated and twelve died of internal hemorrhage. The writer has never seen such fatal hemorrhage in cattle from any other cause than sweet clover. When the skin is removed, one is impressed with the pale, anemic appearance of the tissues. The vessels are empty, for the animals have literally bled to death.

THE BLOOD-VESSELS

A careful study of a large number of sections of the aorta and arteries, as well as of the vessels in the tissues involved in an area of hemorrhage, has been made. It is obvious from the extent and magnitude of the hemorrhage which may proceed to fatal termination within four or five hours that appreciable rents in the vessels actually occur. It is doubtful, indeed, if such hemorrhages are by diapedesis. No visible openings have been found in the vessels although careful search has been made to determine their gross presence. To find minute openings in the walls of the smaller vessels presents very decided difficulties of microscopic technic. It is probable, however, that hemorrhage occurs in the small arteriole bed rather than from the capillaries, veins, or from the larger arteries.

No evidence whatsoever of atheroma, thrombosis, arteriosclerosis, or other injury to the vessel walls has been found. There is no recognizable necrosis or cellular reaction in or about the vessel walls, although we are entitled to suspect a toxic injury as an explanation. It is impossible for the present, therefore, to demonstrate actual injury to the vascular bed whereby the hemorrhage originates, although it should be assumed. Neither is it possible to explain the exciting cause of most internal hemorrhages although parturition, occasional cases of mechanical trauma, castration, and dehorning are more readily understood. The character of the blood renders the further progress of the hemorrhage more comprehensible. An attempt was made to produce it by raising the blood-pressure enormously in two sweet-clover calves by the intravenous injection of adrenalin. The experiment failed. Unfortunately, we have no simple, repeatable clinical method of measuring bovine blood-pressure. Some of our experiment calves subject to considerable handling and venous puncture have shown a decided tendency to bleed into

the pharyngeal and cervical region and such hemorrhage often extends into the mediastinum.

CHEMICAL STUDIES OF THE BLOOD

This work is of interest to show the possible interference with the metabolism of animals as a result of the damaged sweet clover and to reflect tissue injury.

Non-protein nitrogen: Determinations have been limited to those for non-protein nitrogen. While values of urea, creatinine and uric acid have their special significance, they are seldom excessive if the non-protein nitrogen is normal. The method of Koch and McMeekin² was employed. Forty-two determinations were made on sixteen animals, which includes both experimental and field cases in various stages of the disease. Some of the animals were likewise examined during the progress of the feeding period. No appreciable deviation in the non-protein nitrogen of the blood was found. One sample showed 49 mg. per 100 cc of blood. Forty-one samples varied from 25 to 43 and averaged 31.3 mg. per 100 cc of blood.

No evidence has been found of nitrogenous retention in this condition. Certainly, no evidence has been secured to support the contention of Schofield that marked injury to the kidney is characteristic of this disease, although it is true that the chronic glomerulonephritis of man which is accompanied by uremia is rare in live stock. The acute tubular injury more properly termed nephrosis usually shows little or no nitrogenous retention. Further consideration of this phase of the problem is made in this histologic discussion of the kidney and in that of the examination of the urine.

Blood sugar: Determinations of blood sugar were made on the same filtrates as reported for non-protein nitrogen. Thirty-five of the samples varied from 40 to 83 mg. per 100 cc of blood. The remaining samples showed somewhat higher values—103, 111, 181, 127, 144, 115 and 212 mg. Little explanation can be advanced for these higher values. It is significant that they were found after the clinical appearance of hemorrhage. The highest value was secured from an animal which was found on necropsy to have a well-defined hemorrhage from the vascular plexus of the right lateral ventricle of the brain. Tatum⁴ found, with his experimental external hemorrhages, that a hyperglycemia regularly occurred and seemed to be correlated with the diminished alkaline reserves.

Acid-base equilibrium: An interesting volume of literature exists on the neutrality regulations of the animal mechanism. Many papers and reviews deal with the various aspects of the problem. This involves the intricate relationship of intake of acid and alkali, of respiration and excretion, whereby the reaction of the blood or the pH, which is a mathematical expression of the ratio of the blood bicarbonate to the carbonic acid, is maintained within the normal physiologic range. Deviations therefrom which constitute abnormalities are associated with alkali administration, persistent vomiting, over-ventilation, diabetes, cardiac decompensation, diminished respiration and impaired renal function.

There seems to be little in the literature to suggest that the acid-base equilibrium is definitely correlated with the coagulation of the blood. Neither are the aforementioned conditions primarily associated with irregularities in coagulation. It is true that diabetes and the uremia of the chronic glomerulonephritis of man which show such pronounced acidosis are rare indeed in animals. On the other hand, the loss of blood from external hemorrhage is usually followed immediately by a decreased alkaline reserve. Some hours later after bleeding, the alkali reserve is found to be above normal, according to Evans³ and Tatum.⁴

Work was started during 1925 but it was limited to gasometric determinations with the Van Slyke blood-pipette. During the spring of 1927, these studies were continued experimentally on cattle with parallel determinations of the blood pH with the McClelland⁵ hydrogen electrode. The normal range for cattle seems to approximate that of the blood of man, which is given by Myers and Booher⁶ as from pH 7.35 to 7.43, with a bicarbonate content of from 55 to 65 volumes per cent. Van Slyke⁷ gives an average of 7.40, varying from 7.30 to 7.50. Blatherwick⁸ reports 22 determinations on cows of 68.3 to 55.1 (av. 61.5) cc of CO₂ bound by 100 cc of plasma, while Robinson and Huffman⁹ report 112 values ranging from 41.4 to 75.8 (av. 59.2) volumes per cent.

Determinations were made on the blood of normal healthy cattle and continued until after the appearance and during the progress of hemorrhage. Four cattle were thus examined during their feeding trial. Occasional samples were similarly secured from other animals so that the data include fourteen cattle. Sixty-one samples of these bovine bloods varied from 53 to 74 volumes per cent of CO₂ which averaged 62.6. Forty-six deter-

minations of the blood pH, most of which had corresponding determinations by the gasometric method, gave values from 7.33 to 7.49, averaging 7.41. These values are no doubt normal. Only one unusual additional value was found, which was of an animal with well-defined hemorrhage. The pH was 7.54 and the bicarbonate 97.4. The respiration was 9 per minute. No diminished alkaline reserves were found to occur with these internal hemorrhages. It seems therefore that the neutrality regulation of the animal body was not disturbed by the feeding of the damaged sweet clover and the characteristic complex which resulted thereby. The delayed coagulability did not involve an alteration of the pH of the sweet clover blood and it further seems to exclude those profound alterations of cellular and organic metabolism which are so correlated.

PATHOLOGIC STUDIES

The heart: The heart is one of the most common locations of hemorrhage, although the extent of loss of blood therefrom has not resulted fatally in my experience. The pericardium is often besprinkled with hemorrhages which vary in size from mere points to areas several cubic centimeters in diameter. They may be few but they are often present in immense numbers, so that their bright red appearance is a striking picture. The surfaces are smooth and shining. The fluid in the pericardial sac is usually normal. The hemorrhages seem to be confined to the surface and seldom extend into the muscles. The endocardium of the left ventricle is usually blood-stained. No gross changes have been recognized in the myocardium.

Schofield reports finding small areas of necrosis. I have failed, in a careful study of a series of twenty animals, to find such changes. The muscle nuclei and striations are clearly visible. There is no fibrosis nor increase of leucocytes. The sections from most of the animals have shown an infestation with *Sarcocystis blanchardi*. The protozoa seem confined to the heart. Most of our animals were purchased in the vicinity of Fargo and ranged in age from six to eighteen months. They were usually examined during the winter and spring.

The pancreas and adrenals seem to be normal and free of lesions. There is often hemorrhage beneath the peritoneum of the diaphragm.

Lung: There is seldom any excess fluid in the thoracic cavity of cattle although the wall is a common site for large hemorrhagic

sugillations. The fat tissues of the mediastinum often show hemorrhages, yet the parietal and visceral pleura are seldom involved. The lungs are pale, well distended, contain little blood and are quite normal microscopically.

The abdominal cavity may contain one to three gallons of bloody fluid. It has more of the characteristics of fluid blood than of an exudate. It usually occurs when there is hemorrhage adjacent to the cavity.

Digestive tract: The esophagus may be surrounded by a large hematoma which extends from the pharynx to the diaphragm. The presence of one or more hemorrhages on the peritoneal surface of the rumen is almost a constant finding. They vary in size from mere pin-points to large irregular areas of perhaps a square foot in extent. They occur in sheep as well as in cattle. Rabbits likewise show a corresponding tendency to present hemorrhages beneath the peritoneal covering of the stomach. Such hemorrhages lie just beneath the peritoneum and consist of a thin layer of extravasated red cells. The other compartments of the stomach are less frequently involved, although the abomasum has been seen with an extensive hemorrhage involving most of the organ and with a thickness of the wall of perhaps an inch. Schofield reports the presence of hemorrhage beneath the peritoneum of the intestine. I have not encountered it. The mucosa of the digestive tract seems to be normal microscopically. Sections of areas involved in the hemorrhage show normal tissue except for the presence of the free-lying red cells. No evidence of inflammation has been found.

Lymph-nodes: They are normal in appearance except when they are adjacent to an area of hemorrhage, when they are swollen and of a deeper pink color. Such nodes show considerable hyperplasia and evidence of phagocytic activity by the presence of many large endothelial cells or clasmotocytes.

The spleen is invariably normal except for the occasional presence of hemorrhage from the adjacent compartment of the stomach which sometimes infiltrates the capsule. Microscopic examination has failed to reveal evidence of increased phagocytic activity and in only about three instances have even appreciable amounts of golden brown hemosiderin been found. The spleen has failed to furnish evidence that this is a hemolytic disease.

Muscle: Many sections of striated muscle from areas of hemorrhage have been examined. No evidence of Zenker's degeneration or injury of the muscle fibres has been found. Striations,

nuclei, and staining properties are normal. The red cells which constitute the visible residue of hemorrhage are invariably intact and this, together with the visible lack of fibrin, organization and pigmentation suggest the recent effusion of blood. Occasionally an increase of leucocytes is observed in the mass of reds, which suggests an attempt at repair. No other evidence of increased leucocytic activity has been found although the more detailed hematologic studies do not indicate a depression of those functions.

Kidney: The gross appearance of the kidneys is normal. If this is a toxic condition, one might reasonably expect to find a pale, acutely swollen kidney in which the cut surface bulges. These nephroses which involve mainly the convoluted tubules are common in toxemias as of pregnancy, eclampsia, infections, and from the administration of tartrates, mercuric chlorid, chromates and iodine. Schofield reports his finding a characteristic nephrosis although he mentions that the gross appearance of the kidneys is normal. It is true that the microscopic examination of sections for nephrosis presents some difficulty of interpretation. Functional tests are perhaps an even better index of injury. Myers¹⁰ states that the blood cholesterol may be increased several fold in nephrosis. He asserts that a cholesterolemia is pathognomonic for nephrosis. Estimations were made of the blood cholesterol by the method of Ling¹¹ on a series of eight animals during the progress of the sweet clover feeding experiment. The determinations were made two or three times a week during the feeding period. No appreciable increase whatever was found over the normal values or that found at the start of the experiment. The amounts varied from 120 to 250 mg. per 100 cc of whole blood. This work did not indicate the presence of a nephrosis.

The glomeruli are normal. I cannot satisfactorily confirm Schofield's observations with reference to the tubules. In a series of sections from more than twenty cattle dead of sweet-clover hemorrhage, I found a few casts in the kidney of but one animal. No deposition of hemosiderin has been found.

Examination of the urine of calves fed sweet clover experimentally was made to ascertain further the possibility and extent of kidney injury. The work was started during 1927 and examinations were made of the urine of seven calves at intervals during the feeding trials. Tests were made for albumen by the heat-acetic acid, Heller and potassium ferrocyanid methods but these

were negative in every instance. Tests were made for hemoglobin by the guaiac method, for sugar by Benedict's, and for bile pigment by Gmelin's and by Kapsinow's methods. These examinations as well as those for casts were negative in every instance.

Tests for albumen were made on another series of ten animals during the spring of 1928 at intervals during the progress of the feeding experiment. An average of six examinations was made per animal. Albumen was found in the urine of only two animals. It occurred in the urine of calf 82 shortly before death from an extensive hemorrhage which surrounded the kidneys. It was present, however, in the urine of calf 81 after being fed for 22 days. It persisted until the death of the calf, ten days later. This is the only instance in which the presence of albumen has been recognized and which might have resulted from a nephrosis. Little has been found, therefore, to indicate regular and serious injury to the excretory function of cattle which are fed on the damaged sweet clover.

Liver: The gross appearance of the liver is usually normal. The size, color and consistency show little recognizable alteration if postmortem changes are positively excluded. The cut surface is likewise uniform in appearance. Hemorrhages up to 2 or 3 cm. in diameter, beneath the capsule or in the parenchyma, have been seen but occasionally, although Schofield reports them as common.

If we assume that the cause of this condition is toxic in character rather than infectious in nature, then since absorption is by way of the digestive tract, one might expect to find lesions in the liver or functional evidence of injury. Conditions which involve a prolongation in the coagulation time of the blood are frequently associated with liver injury. Examples thereof are severe choleo-cystitis and poisoning from chloroform and phosphorus. The first is accompanied with icterus, while the other two conditions show retrograde fatty changes in the liver. Foster and Whipple¹² report that low fibrin yields from the blood are associated with extensive liver injury, while mild injury serves as a stimulation to the formation of increased amounts of fibrinogen. Analytical work on the blood of a series of sweet-clover calves seemed to indicate that there may be some depression in the blood fibrinogen during the feeding trial, yet the values obtained were within the probable error of a series of determinations on normal cattle.

The presence of areas of focal necrosis seems to be the only significant microscopic finding. This injury does not seem to be constant, for it has been recognized in only about one-half of the cases, which include more than twenty animals, most of which were cattle. These areas may occur any place in the lobule and three or four of them may occur in a low-power field.

Schofield reports that whole lobules may be destroyed but I believe this to be evidence of postmortem changes. The cytoplasm of the focal areas of necrosis is granular, stains poorly with eosin, is often "muddy," while the nuclei are pyknotic. There is little evidence of cellular reaction. A series of nineteen livers from cattle, sheep and rabbits dead of sweet-clover disease were examined for retrograde fatty changes but none was found except in three of them.

Physiologic studies indicate that a gradual progressive diminution in the prothrombin of the blood is the explanation for the delayed coagulability of the blood. Schofield attached more significance to his findings of liver injury than to the alteration in the blood. The long period of delayed coagulability of the blood, sometimes four to six weeks, is not consistent with the extent of visible injury to the liver which has a high factor of safety. The prompt recovery of many treated cases and the general lack of any indication of intoxication would lead one to conclude that no serious organic functional changes in the vital viscera are concerned.

The liberation of hemoglobin is regularly followed by an increased production of bile pigment. This occurs with such hemolytic diseases as malaria, Texas fever, anaplasmosis and septicemia. Schofield emphasizes an anemic phase of the disease and assumes a destruction of the red cells. It is not my purpose to discuss the hematologic phase of the disease at this time. I do not believe that death ever results in cattle from hemolysis or loss of red cells except they leave the vascular bed by hemorrhages. In severe hemolytic anemias, the hemosiderin is quickly deposited in the spleen, liver and kidneys. I have never found it in this disease in either the liver or kidneys. Clinical icterus has never been recognized in our series of about 75 experiment animals.

The icteric index of the blood plasma has been determined at regular intervals during the feeding experiment by the method of Rockwood and Szczypinski¹³ on a series of ten experiment cattle. No increase in the bilirubin in the blood was found

except in one animal, during recovery from hemorrhage, when it increased about four fold. Nothing was found, therefore, to suggest an increased destruction of blood prior to hemorrhage.

SUMMARY

This paper deals with some of the pathologic and functional features of the interesting hemorrhagic complex which results from the feeding of damaged sweet clover hay and silage.

Death in cattle seems to result only from hemorrhage, which is preceded by the constant development of the delay in the coagulability of the blood. No visible alteration has as yet been found in the blood-vessels to explain the internal hemorrhagic diathesis.

No retention of non-protein nitrogen of the blood has been found. The blood sugar is regularly normal although a few determinations which were high were found. Such values were occasionally found to accompany hemorrhage.

Studies of the neutrality regulation of the animal's blood with the hydrogen electrode and the gasometric pipette showed no disturbance of that intricate mechanism.

An infestation of the heart muscle of our experiment calves with *Sarcocystis blanchardi* has been an interesting and almost constant incidental parasitologic finding.

The absence of albuminuria and cholesterolemia, together with the normal gross and microscopic appearance of the kidneys, indicate the absence of toxic effects thereon.

Focal necrosis is a common lesion in the liver. There is a question however if it is definitely correlated to the delay in the coagulation of the blood. Little evidence has as yet been secured to show that the function of the liver is seriously impaired. The absence of hemoglobinuria and icterus and the low icteric index, together with the failure to find deposition of hemosiderin, indicate that this is not a hemolytic disease.

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DISCUSSION

DR. L. W. Goss: I have been interested in this work Dr. Roderick has done. I think he has made a very thorough search for lesions, but I wonder, from the hemorrhages I have observed during the past year, if these are not through the capillary bed rather than through the larger vessels. Adrenalin chlorid is a vaso-constrictor. In this case hemorrhage might not occur with constriction of the vessels. It might be, if laudanum had been used, that other results would have been secured. I wonder if that has been tried?

DR. RODERICK: It may be that some of the smaller hemorrhages may be through the capillaries. I am inclined to believe that the more serious hemorrhages are from the larger vessels. Now one of the slides that I showed was that of an animal which probably bled to death in the matter of a few minutes, or half an hour. I have seen an animal bleed to death, quite a number of them, in two or three hours, so I am inclined to think vessels of fair size are involved. I have not tried laudanum.

Women Veterinarians

The neglect of an efficient veterinary service in any country may have appalling consequences, as was shown by what happened in Russia. After the revolution veterinary surgeons, like most other educated people, suffered, and the veterinary service passed into the hand of a cavalry officer. Within a year many of the most destructive of animal diseases were epidemic throughout the land.

For years the United States has had a competent, although not always adequate, veterinary service, one of the most interesting developments of which has been the admission of women to practice as veterinary surgeons.

It is pointed out that, while natural chivalry made men shudder when they thought of women doing the oftentimes unpleasant work which accompanies medical and surgical attendance on sick cows and horses, there is an enormous field before them in connection with the smaller animals, such as dogs, cats, rabbits and birds. Women, it is urged, have more gentle hands than men, and show more affectionate care for domesticated animals, and these qualities would go far toward making them ideal animal doctors.

Experience proves that professional women are nothing if not thorough, so one may easily feel confident that no part of their subject will be neglected, and that the horses and cattle will receive from them their proper share of sympathetic and expert attention.

—Owensboro (Ky.) Messenger.

BREEDING DISEASES OF THE HORSE*

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It is a well-known fact that breeders of live stock seldom experience the rare good fortune to have 100 per cent of pregnancies among the females maintained for breeding in any one season. To have all females of breeding age, of any breed or species, in a given establishment, give birth to healthy offspring at regular seasonal intervals would be the ideal. With a problem surrounded by and dependent upon so many vital phenomena, this high point in breeding efficiency is seldom reached. It is, nevertheless, the goal toward which all breeders are striving. The regular seasonal reproduction by females (cattle, swine and horses) is first of all an important economic factor. The failure of an individual to reproduce its kind at regular intervals is, in addition to the economic loss, a serious disorganization of plans for constructive breeding; and, further, in cases of permanent sterility the loss of valuable blood lines may result. It is realized that most veterinarians and many breeders know full well the economic losses in animal breeding that come from sterility and that they are in general equally aware of the many diseased and abnormal conditions of the reproductive organs that result in sterility.

Our work on breeding diseases of horses has been very largely with the light type of animal. Mention is made of this fact because many of you may have had experience with the heavy type of horse. From our limited experience with the heavier breeds it is not possible to say that what has been found to be the basis of trouble in the lighter types is equally true for the draft horse. We do know that in some cases the cause of the trouble is the same for both.

During the past ten years the raising of Thoroughbred horses has been unusually profitable. Naturally the breeder would be especially concerned as to the number of his brood mares that conceived and that produced *live, healthy foals*. The breeder has always been aware that sterility in mares was one of his major problems. Veterinarians have known for a long time the general nature of disease of the reproductive organs of brood mares.

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

The problem was to make a detailed investigation of the causes of sterility, and develop improved and accurate methods of diagnosis. The classification of non-breeding mares on the basis of the cause is essential before special forms of treatment can be started. With assurance of the cooperation of both breeders and practicing veterinarians, an investigation was undertaken.

BACTERIOLOGICAL EXAMINATION ESSENTIAL

Early in the work we found that infection of the genital tract, which results in cervicitis and metritis, was the most serious form of sterility. In the diagnosis work it was necessary to distinguish between non-infected and infected mares and determine the character of the infection if present. The only way to do this was by the employment of bacteriological methods. Therefore, we have always emphasized the importance of a technically correct bacteriological examination as a routine part of the clinical examination. It may be possible on clinical examination, especially for one who is familiar with the normal genital tract of the mare, to distinguish between infected and non-infected mares in a high percentage of cases. There are many cases, however, in which it is absolutely impossible to determine the presence or absence of infection in the cervix and uterus unless one resorts to a bacteriological examination.

While our project was primarily on sterility in mares, it included impotency in stallions, diseases of foals, and abortion as related to disease and infection of the genital tract of mares and stallions. The material of this report is a result of the work of eight or nine years.

In case of disease of the reproductive organs, the report is based on the examination of 2148 non-breeding mares. Included also are the examinations of 161 fetuses, 125 foals and 61 stallions.

In the 2148 mares are included only those mares studied in which the clinical examination was supplemented by the inoculation of two agar slants from the cervix and uterus, the tubes being incubated and examined before a diagnosis was made. Many other mares were examined but are not included in this report, because no bacteriological examination was made of the reproductive organs. Culture tubes were inoculated from the genital tract of some of the mares only once, from others many times. Altogether, about 5000 bacteriological examinations were made of the living mares, or an average of about two and one-half to the animal.

In those breeding establishments where the brood mares are dealt with according to the general plan which was developed in connection with and on the basis of our studies, procedure is as follows: All brood mares bred are examined for pregnancy from two to four months following the close of the breeding season. The examinations for pregnancy are usually made in September and October, although each year there is a growing demand to have the mares examined much earlier. Now many mares are examined from twenty-one days and up following the last breeding date. As soon as the pregnancy examinations have been made, the pregnant and non-pregnant mares are separated. The pregnant mares are kept in fields and stables especially prepared for them and are otherwise handled and cared for by a systematic and carefully executed method.

MARES ARE CLASSIFIED

In handling, studying and planning for treatment of the barren mares, in the field, they are first divided into two main clinical groups. Group 1—infected mares. Group 2—non-infected mares. The infected mares are further classified on the basis of the type of infection found on bacteriological examination, as follows:

Group 1a—mares infected with *Streptococcus genitalium*.

Group 1b—mares infected with *Encapsulatus genitalium*.

Group 1c—mares infected with miscellaneous microorganisms.

The non-infected mares are further classified into five divisions on the basis of clinical examination only. This classification is essential if the mares are to receive attention before the beginning of the next breeding season. In most of our work we have found it satisfactory and convenient to classify the non-infected barren mares as follows:

Group 2a—mares apparently normal.

Group 2b—mares with abnormally large, cystic and diseased ovaries.

Group 2c—mares with abnormally small, hard ovaries.

Group 2d—mares physiologically exhausted.

Group 2e—mares with uterine and ovarian tumors and non-infected degenerative changes in the uterus.

Of the 2148 mares examined, it was found, on the basis of our method of examination, that 1411 (65.68%) were apparently free from infection of the genital tract and that their failure to conceive was due to causes other than infection. Seven hundred

thirty-seven (34.31%) were found to be suffering with cervicitis and metritis due to different types of infection. The percentage of infected mares as compared with non-infected has dropped from over 40 per cent, found in the first years of the work, to 34 per cent at present. This is due to several factors. In the beginning only the barren mares suspected of being infected were examined and cultured. Later all barren mares on the farm were examined. For the last three years many stallion-owners have required a health certificate for all barren mares before admitting them to service. The certificate must state that the mare has been examined and cultured (cervix and uterus) and shows no clinical evidence of inflammation of the genital tract, with cultures negative to bacterial growth.

BACTERIOLOGICAL FINDINGS

Of the 737 mares classed as suffering with genital infection, it was found that 509 (69.1%) were infected with *Streptococcus genitalium*, and 65 (8.8%) were infected with *Encapsulatus genitalium*; while 163 (22.1%) showed miscellaneous infection. The mares classed as non-infected yielded negative bacteriological examinations each time they were cultured and were otherwise apparently normal and free from infection of the genital tract. The mares classed as infected yielded positive results upon bacteriological examinations, on one or more times that cultures were taken, and also showed clinical evidence of cervicitis and metritis. We have been unable to keep in touch with all the mares examined. Those that we have been able to follow since the first examination was made have, with few exceptions, come out almost exactly according to the prognosis made on the basis of the results of the examination.

Altogether we have had for study 161 equine fetuses. All of these were examined postmortem and studied bacteriologically. There seems to be no satisfactory way to classify these cases of abortion except on the basis of the bacteriological findings.

Of the 161 fetuses examined, 89 were negative; from 55, streptococci were isolated; from 5, staphylococci; from 10, *Bacillus abortivo-equinus*; from one, *Bacterium viscosum equi*; and, in the case of one, postmortem decomposition was so bad that no diagnosis could be made. Of the 89 negative cases of abortion, 14 were twins; one was apparently due to anatomical abnormality of the fetus and one was removed from a mare that died suddenly. If these 16 cases are excluded, we have a total of 145, of which 73

(50.3%) were negative and 72 (49.7%) were positive on bacteriological examination, streptococcic infection predominating.

The small number of fetuses showing infection of *Bacillus abortivo-equinus* is due to the fact that the mares on practically all the farms from which the fetuses were obtained had been vaccinated against equine contagious abortion. None of the fetuses showing abortion infection were from vaccinated mares. During the last nine years there have been only three or four horse-breeding farms on which equine contagious abortion occurred in a serious form and in no case were we able to secure for examination all the fetuses that were aborted. Most cases of abortion in which the fetus was negative on bacteriological examination have been isolated cases occurring here and there on many different farms. We have, however, in a few instances had as many as eight or ten fetuses from the same farm, all abortions occurring within a short time and all proving negative on examination.

NUTRITIONAL DISTURBANCES CAUSE ABORTION

No anatomical or special pathological changes were observed in the fetuses and the mares returned to normal without treatment. We have been unable to determine the cause of abortion in these cases. There was in every case, however, some evidence to suggest that possibly the abortions were the result of nutritional disturbances in the dam. Another possibility is that they were due to some form of infection with which we are not familiar and which fails to grow on the ordinary culture media.

Our study of foals is most interesting. Most of them were dead at birth or died shortly after birth. A few were premature but lived for some time and a small number were several weeks or months of age at time of death. Altogether, 125 have been examined. As in the case of the fetuses, the foals are classified largely on the basis of the bacteriological findings. Of the 125 examined, 31 were negative. In the case of 10 of these, death was due to traumatic injury and dystokia; from 6, *Bacillus abortivo-equinus* was isolated; from 47, *Bacterium viscosum equi*; from 25, streptococci; from 7, *Bacterium coli*; 8, miscellaneous; and in 2, postmortem decomposition prevented an accurate diagnosis.

The examination showed that the large majority of deaths were due to infection, *Bacterium viscosum equi* being responsible for the death of a larger number of foals than any other single micro-organism, streptococcic infection coming second. With very few exceptions it was apparent that the infection present was of pre-

natal origin. In the case of streptococcic infection the micro-organism was presumably either present in the genital tract of the mare when bred or was introduced at the time of service. In the case of foals dying with infection of *Bacterium viscosum equi* of prenatal origin, we have never been able to determine just how or when the infection enters the uterus of the dam. We have under way at present breeding experiments that we hope will throw some new light on the channels of infection for this organism.

POTENCY OF THE STALLION

In the study of breeding problems in mares the potency of the stallion is a most important factor. During the spring of 1926 and 1927, the semen of 61 stallions was examined. A few of these stallions had been examined in June, 1925, and a few were examined in 1928. An examination of the semen consisted of estimating the percentage of motile cells, determining the hydrogen-ion concentration by the colorimetric method and a study of the cells in fixed and stained smears. In about 50 per cent of the stallions examined, there was a direct correlation between motility, reaction, morphology of cells and the potency of the stallion. In these stallions the motility ranged from 80 to 95 per cent; the reaction from pH 7.3 to pH 7.7, and approximately 75 per cent of the cells in each case were estimated as normal in the fixed and stained smears. Further, a high percentage of the mares bred to these stallions produced foals.

In the balance of the stallions examined there was an apparent lack of direct correlation, although many were potent. In the case of a few young stallions, first year in stud, and a few old stallions, one would not have hesitated to state, on the basis of the semen examined, that they would prove impotent as there were very few cells in the semen and all were non-motile. On the other hand we have encountered stallions that would have been pronounced highly potent, on the basis of the semen examination, that were absolutely impotent on the basis of their breeding record.

As a result of our work with stallions we have come to feel that in the semen examination, motility, including percentage, degree and duration is, if correctly estimated, the best criterion of all upon which to estimate the potency of the stallion. However, there is an occasional exception and in the final analysis potency or impotency are to be determined by the breeding record.

The condition of the mares bred to the stallion should always be taken into consideration.

In some stallions it was found that the percentage of motility was low but the degree of motility in individual cells was good and the horse proved in service to be highly potent. In other cases the percentage of motility was fairly high, yet the degree of motility of individual cells was very low and the stallion was actually low in potency, as determined by the breeding record.

The reaction of semen is found to be more alkaline in the older stallion (pH 7.7 to 8.0) than in the young stallion (pH 7.3 to 7.6). In only three cases have we found a sample of semen that was less alkaline than pH 7.2, the average being pH 7.55.

The consistency or physical properties of the semen is suggestive, if not actually indicative of the potency of stallions. In the old stallions the semen has a tendency to become thin and watery. In young stallions which were important the seminal fluid was usually observed to be thin and watery. From an examination of the semen of stallions much valuable information can be obtained; the results of the examination usually correlate directly with the actual potency of the horse. Therefore, before drawing definite conclusions as to the potency or impotency of a stallion, his breeding record, past and present, must be taken into consideration. The care, management, and handling are, on the basis of our observations, most important. A number of stallions that were impregnating less than 50 per cent of the mares bred to them were, with careful handling, feeding and treatment, greatly improved.

I wish to make mention of the fact that Dr. P. R. Edwards, bacteriologist, has done most of the bacteriological work in connection with this study; and that Dr. F. E. Hull, assistant veterinarian, has made many of the semen examinations.

SUMMARY

1. It has been found that sterility or barrenness in mares, in approximately one-third of the animals examined, was due to infection. The predominating organism in cases of cervicitis and metritis is a streptococcus. In a smaller number of cases encapsulated bacilli have been found to be the etiological agent of severe infections of the genital tract.
2. Streptococci live as saprophytes on the external genitals of mares and stallions.

3. Streptococci are found just inside the lips of the vulva in maiden mares and in mares in foal.
4. One-half of all mares foaling normally, that were examined bacteriologically, showed streptococci in uterine secretions from one to nine days after foaling.
5. Most of the foaling mares showing streptococci in the uterine secretion after foaling will return to normal without treatment.
6. Streptococcic infection of the genital tract seldom takes place except through the development of predisposing influences. A small percentage of foaling mares showing no clinical evidence of disease became infected following parturition and from service.
7. The principal predisposing causes to uterine infection in foaling mares are: dystocia and lacerations and bruises along the genital tract at foaling time.
8. Barren mares, free from infection of the genital tract, are more resistant to infection at time of service than are foaling mares.
9. Cervicitis and metritis in mares, due to encapsulated bacilli, while not common as compared with other types of infection, is most serious when present.
10. The prevention of streptococci and other forms of infection in the genital tract of mares is fundamentally a question of sanitation and breeding hygiene.
11. Most mares suffering from uterine infection will fail to conceive when bred.
12. It has been determined that encapsulated bacilli causing metritis in mares are readily transmitted from mare to mare by the stallion at the time of service, the greater danger being the first few days after serving an infected mare.
13. Stallions do not become permanent carriers of this micro-organism.
14. Mares harboring infection in the genital tract, or that are infected at the time of service, if they conceive, will frequently abort.
15. Streptococci are found in a large percentage of aborted fetuses.
16. Colts carried full time by mares with streptococcic infection are often stillborn, die at birth, or die within the first few days or weeks from streptococcic infection.

17. *Streptococcus genitalium* is often the principal infecting agent in navel-ill, joint-ill, peritonitis, and septicemia in very young foals.
18. Other infections found in stillbirth and diseased foals are *Bacillus abortivo-equinus*, *Bacterium viscosum equi*, and miscellaneous infections, such as *Bact. coli*, staphylococci, etc.
19. *Bacterium viscosum equi* has been found to be the cause of death in one-half of all foals examined.
20. The large majority of these cases of infection in young foals are unmistakably of prenatal origin.
21. The principal distinguishing clinical feature between cervicitis and metritis in mares, due to streptococci and encapsulated bacilli, is the character of the exudate.
22. A bacteriological examination of the cervix and uterus of mares as used in this study, if properly carried out, is a most reliable method of determining the presence or absence of infection in the genital tract of mares and is the only method by which a positive differential diagnosis can be made.
23. Many barren mares are consistently negative on culture; most infected mares are consistently positive on culture.
24. Mares showing clinical evidence of cervicitis and metritis, even if the cultures from them are negative, should be re-cultured.
25. Mares showing no evidence of inflammation in the genital tract, but showing positive on cultures, should be re-cultured.

Errata

Dr. E. A. Watson directs attention to several errors made in compiling averages from data used in preparing the manuscript of the paper, "Researches on *Bacillus-Calmette-Guérin* and Experimental Vaccination Against Bovine Tuberculosis," by E. A. Watson, C. W. McIntosh and H. Konst, published in the November, 1928, issue of the JOURNAL (lxxiii, n. s. 26, No. 7, pp. 799-816).

Page 807, line 5—94 (guinea pigs) should be 92.

192 (days) should be 295.

line 6—32 (guinea pigs) should be 30.

line 8—9 (guinea pigs) should be 12.

Page 809 (Table I), line "Average duration in days," column "Killed," 365 should be 374; column "No Lesions Discernible," 192 should be 295.

THE RELATION OF DIET TO DISEASE*

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For many centuries clinical evidence supported the generally accepted view that diet plays an important role in the prevention and alleviation of disease. But it has not been until quite recently that experimental evidence was sought to prove this hypothesis. Even at the present time, the experimental data are of a fragmentary and contradictory nature.

In attempting to study the influence of diet on the resistance to disease, one must deal with a vast field and many perplexing ramifications.

Diet plays an important role in the treatment of such diseases as diabetes, nephritis, high blood-pressure, gastritis, gastrointestinal disorders, fevers, constipation, and any other disturbances of the normal metabolic processes. Here we have an abnormal mechanism so that the body is unable to utilize properly a normally adequate diet. In such conditions, the foods should be so selected that they will not only furnish an adequate supply of all the dietary essentials, but they must be furnished in a form which the depressed organs can digest, assimilate and utilize. On the other hand, even in a normal individual, similar deficiencies result when an otherwise adequate diet is fed in an undigestible form.

Every adequate diet must contain sufficient amounts of fat and carbohydrate to furnish the calorific needs, a sufficient amount of protein, chemically able to furnish all the essential amino-acids, a supply of minerals able to furnish all the inorganic constituents of the body, and an ample supply of each of the vitamins. Since the carbohydrates are utilized only to furnish energy, the quality is essential only in so far as their digestibility is concerned. Enough carbohydrate must be present, however, to aid in the oxidation of fats. When the amount drops below this minimum, fats are oxidized only to the four-carbon compounds such as acetone bodies. The fats, however, have a calorific value and also furnish some of the vitamins and hence should be selected as a source of these dietary essentials.

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

The proteins are, from a nutritional viewpoint, not definite chemical entities, but merely composed of many amino-acids. Although our knowledge of the amino-acids is still incomplete, experimental evidence has shown that certain of the amino-acids are essential for the normal growth and functioning of the body. In this group may be mentioned tyrosine, tryptophane, histidine, lysine, cystine and cystein. Of these, some act in maintaining growth, others by furnishing material for the production of essential secretions. Thus, tyrosine has been shown to be structurally and metabolically related to thyroxin and adrenalin. Cystein is essential for the production of glutathione, which is closely associated with the oxidative processes in the body. Histidine is structurally related to histamine, whose action on the capillaries is so marked.

QUALITY OF PROTEIN IMPORTANT

Since the various proteins do not contain all the essential amino-acids, it is obvious that the old practice of furnishing a certain amount of protein without considering its quality is a dangerous one that may lead to disastrous results. Some proteins lack one or two essential amino-acids but contain an abundant supply of all the others, and hence should be supplemented with other proteins containing these. We must furnish all the essential amino-acids in adequate amounts to insure normal growth and well-being and the protein part of the diet must be selected with this in view.

Since the body is unable to manufacture or create any element, it is of prime importance to furnish, either in the food or water, all the inorganic elements which are found in the body structures or fluids. These may be used in the formation of the endocrine secretions, digestive ferments, the formation of the skeleton and softer parts of the body, for growth of tissues, and in regulating such metabolic processes as the maintenance of a constant acid-base equilibrium, osmosis, diffusion, formation of lymph, and thus the nourishment of all tissues.

Minerals must be supplied not as so much ash, but each inorganic substance must be considered separately. We have but to think of the close relationship between the amount of iron in the diet and the production of anemia, the amount of calcium or phosphorus and such diseases as rickets, osteomalacia, abnormal dentition and milk fever, and between the amount of iodine and goitre or hairless newborn.

Even though a mineral substance may be normally found in only very minute traces, the lack of these minute amounts may cause very severe symptoms. Only mere traces of iodine are normally found in the body, but when this minute amount is lacking very severe symptoms develop. Although this is primarily a disease of the thyroid of the dam, the symptoms are manifest chiefly in the offspring. The dam usually exhibits no very striking symptoms except, in some cases, an enlarged thyroid gland. The offspring, however, are born hairless, weak, and oftentimes dead. Pigs and sheep are very commonly affected and calves and colts more rarely. The daily administration of iodids during the period of gestation prevents the onset of this disease. For pigs, one grain of potassium iodid daily during the period of gestation has proven efficacious and for other animals proportionate amounts.

PICA, OSTEOMALACIA AND LOIN DISEASE

The partial deprivation or absolute lack of either calcium or phosphorus produces symptoms which may be manifest in any of our wild or domesticated animals. In cattle the lack of either calcium or phosphorus is followed by a group of characteristic symptoms such as weak muscle tonus, abnormal gait, stiffness, lameness, enlarged, painful joints, delayed development of the teeth, perverted appetite, and marked digestive disturbances. There is a persistence of cartilage in the uncalcified condition. This condition is quite prevalent and, although known by various names, is undoubtedly the same malady resulting from a mineral deficiency. Theiler described it in South Africa as pica; Tuff, in Norway, as osteomalacia; and a similar disease affecting large numbers of cattle in Minnesota and Texas is called loin disease.

The outstanding symptoms are a perverted appetite and general muscular weakness. There is a marked craving for such things as bones, wood, stones and earth and it is not uncommon to see the affected cows eating the skeletal parts of an animal which had recently died of this malady. It occurs most commonly in early spring when the grass is first becoming plentiful. The cows, strong, healthy and fat, at first rest more than normally and within twenty-four hours are unable to rise. If these animals are left to care for themselves, they die within two or three days; but if given shelter, shade, water and food, and frequently turned over, they may live for several weeks. Carefully controlled experiments have led to the conclusion that the cause of this

disease is twofold. The lack of either calcium or phosphorus in the diet causes a perverted appetite. The affected cattle crave mineral and therefore eat the bones of animals which have died of loin disease. These bones contain a toxin which is the primary cause of the paralytic symptoms.

In localities where loin disease is prevalent, the soil is found to contain a lack of calcium and phosphorus and hence the plants are likewise deficient in these elements. It is not surprising, therefore, that the animals eating these plants should show deficiency symptoms. Supplying sterilized bone meal prevents the occurrence of this disease.

A similar condition affects pigs and is known as posterior paralysis, leg weakness, loin weakness or rheumatism. It is associated with a deficiency of either calcium or phosphorus and is characteristically rickets. The affected pigs become sluggish, present a marked lassitude, later a characteristic jerky gait, muscular weakness, knuckling over, painful and often enlarged joints, and curvature of the long bones, especially of the limbs. Experiments have shown that supplying calcium and phosphorus in proportions as low as two per cent of the rations prevents and cures this malady.

RICKETS AND ULTRAVIOLET LIGHT

Dogs also are very susceptible to rickets, especially during their period of growth. Although rickets is primarily a disease resulting from the lack of calcium and phosphorus in proper proportions, in its treatment several factors must be considered. It is obvious, of course, that first we must furnish the lacking minerals in a form which can be utilized by the body. Then we must enable the body to absorb and assimilate these elements. It has been shown that ultraviolet light, present in sunlight, enables the body better to utilize its inorganic constituents. When this light is absent, rickets results. However, vitamin D, which will be discussed later, is able to replace sunlight.

Any other factors, the most common of which is intestinal parasitism, may so interfere with the normal metabolic processes as to prevent the proper assimilation and utilization of the inorganic, as well as the organic, constituents of the diet.

Thus, in the treatment of rickets, in either man or animal, we must first remove any factor which interferes with the normal mechanism. Then, by supplying vitamin D in the form of cod-liver oil, exposing the animals to direct sunlight, and furnishing an

adequate amount of the essential inorganic constituents, rickets can be prevented or cured.

Iron is an important constituent of hemoglobin and is therefore a dietary essential. Recently it has been shown that a minute amount of copper added to the diet enables the body better to utilize its iron. Copper and iron together are much more efficacious in the treatment of nutritional anemias than is iron alone. Liver ash is also very effective in the treatment of this condition.

It is thus seen that fats, carbohydrates, amino-acids, and the inorganic constituents each have an important function in the body. However, recent discoveries have shown that, although chemical analyses of foods furnish some valuable information in regard to their nutritional value, biological assays are final proof of their efficiency. It has been known for centuries that diet was closely associated with such diseases as scurvy, beri-beri, rickets, pellagra and keratomalacia. It was not until recently, however, that experimental evidence proved that these diseases are due to the lack of specific substances in the foods. Although the general term vitamin has been given to these dietary essentials, they have been known also as growth-promoting substances, food accessory factors and food hormones.

SIX KNOWN VITAMINS

Classified according to their activity, there are at present six known vitamins:

- Vitamin A, the lack of which produces xerophthalmia,
- Vitamin B, the lack of which produces beri-beri and polyneuritis.
- Vitamin C, the lack of which produces scurvy.
- Vitamin D, the lack of which produces rickets.
- Vitamin E, the lack of which produces sterility.
- Vitamin P-P, the lack of which produces pellagra in man and sore-mouth in dogs.

There are certain symptoms common to all the avitaminoses such as failure of young animals to grow, failure of mature individuals to maintain their own weight, and general cachexia. But aside from these, the lack of each vitamin produces a characteristic and definite syndrome.

The lack of vitamin A is associated with a definite xerophthalmia. This is caused by secondary invading organisms which gain entrance after the lachrymal secretions are inhibited and there is a drying of the cornea. The sebaceous glands also fail to function

and hence the skin becomes dry and the coat rough. The digestive secretions suffer similarly because of atrophy of the glands and consequently gastro-intestinal disorders are common. A profound atrophy and necrosis of the villi enables organisms which are normally found in small numbers in the intestine to increase and invade the tissues. They are often found in dense masses in the lumen of the glands.

In poultry the lack of vitamin A produces nutritional roup. The symptoms are characteristic. A discharge from the nostrils, which is at first watery but soon becomes viscid, may collect in the infraorbital sinuses where it is transformed into a caseous mass. On the mucous membranes of the mouth, pharynx, esophagus and crop there appear small, white, caseous patches. The renal tubules are filled with urates giving the kidneys a white, streaked appearance. In many of the birds xerophthalmia develops. This is manifest at first as a slight hyperemia of the conjunctiva and a profuse watery secretion, but it soon becomes viscid and is transformed into a large caseous mass. Furnishing vitamin A in the form of fresh green food or cod-liver oil prevents and cures this malady.

VITAMIN B ABUNDANT IN YEAST

Vitamin B is associated with the activity of the muscular and nervous systems. A lack of this substance in the diet is followed by disturbances of the coordinating power of the muscles, a polyneuritis with its attendant paralysis, a characteristic loss of appetite, and degeneration and atrophy of many of the parenchymatous organs. Experimentally this deficiency has been produced in pigeons, rats and dogs, but it is not frequently seen because of the wide distribution of vitamin B. A very concentrated and readily available source of this vitamin is yeast.

Vitamin C, the lack of which is followed by scurvy in man, does not seem to be essential for most domestic animals.

Vitamin D is a fat-soluble vitamin which prevents or cures rickets when an adequate mineral supply is available. Since direct sunlight may replace vitamin D as an antirachitic agent, most of the cases of rickets in our domestic animals are probably due, not to a lack of this factor, but rather to a lack of the proper amount of the essential mineral elements. However, in dogs kept indoors, the lack of this vitamin may become apparent.

In chickens, the lack of vitamin D causes great losses and the resulting malady is known as leg-weakness. It is manifest usu-

ally about four to six weeks after hatching, although much older birds may become affected when raised in confinement. The birds appear weak, the comb and wattles are pale and there is at first an unsteady gait, followed by an inability to stand. The characteristic posture is with the head drooping and hocks on the ground. Cod-liver oil is a concentrated source of this vitamin.

Vitamin E, the lack of which produces sterility, is of great importance for the development of the embryo. If vitamin E is lacking, estrum, ovulation, coition, and implantation take place normally. The affected animals appear healthy and normal and show no outward symptoms of the deficiency. The embryos are normal at first but soon there occurs retardation of development and resorption. The maternal portion of the placenta continues to live for some time afterward and hence it seems that it is the embryo which requires vitamin E. A careful study of this vitamin may aid in the solution of many problems dealing with sterility, abortion, and mummified fetuses. A good source of this vitamin is wheat-germ oil, although other vegetable oils contain it.

VITAMIN P-P AND BLACK TONGUE

Vitamin P-P is the factor which prevents the onset of pellagra. It has recently been shown by various investigators that what was previously called vitamin B is multiple in nature. Two of these factors have been indentified by their activity, namely the antineuritic B and the vitamin P-P (pellagra-preventing). Factor P-P is associated with the prevention of black tongue or sore mouth in dogs. By withholding this vitamin, sore mouth becomes manifest and by supplying the factor P-P, this malady may be prevented or cured.

It has thus been shown that a lack of vitamins exercises a marked specificity in its effects; that the lack of a definite vitamin produces a definite syndrome and that this syndrome may be prevented or cured by furnishing the lacking vitamin.

However, the most common cause of lowered vitality is not the complete absence of one or more dietary essentials, resulting in a prompt appearance of a spectacular deficiency disease, but rather those milder deficiencies which gradually lower the general vitality of the individual without causing any striking symptoms. It is these milder forms of malnutrition which are responsible for so many of the abnormalities for which we have formerly been unable to offer an explanation. Some of the many symptoms

which such chronic, mild, mixed deficiencies may cause are lack of growth, lack of physical development, instability of the muscular and the nervous systems, gastro-intestinal disorders, marked loss of recuperative power and endurance, and especially important is the diminished resistance to infection and toxins. Clinical experience indicates quite conclusively that malnutrition renders the affected animal much more susceptible to infection, toxins and poisons.

DIET AND SUSCEPTIBILITY TO TUBERCULOSIS

Many investigators have shown that there is a close relationship between diet, especially vitamin A, and the susceptibility to tuberculosis. Others have demonstrated that dietary deficiencies render the animals more susceptible to cancer, leprosy, sinus infections, respiratory infections and bacterial invasion in general. Experiments performed by the writer, as well as by others, have demonstrated conclusively that rats suffering from the effects of dietary deficiencies are more susceptible to bacterial infections and succumb to milder attacks of the resulting infection than do normal animals. We also found that white rats, fed a diet deficient in vitamins A and D, B, or E, succumbed to much smaller doses of tetanus toxin than did the normal controls. It required from forty to one hundred times as much toxin per gram body weight to kill a normal rat as it did to cause the death of an avitaminic one.

In order to determine the cause of this cataphylaxis, an attempt was made to study the effect of avitaminosis on the production of antibodies. The results indicate that the avitaminic animals responded to bacterial injections with a much diminished formation of agglutinins and exercised markedly less bacteriolytic power than did the normal controls. The details of these experiments will be reported elsewhere.

After this discussion of the effects produced by dietary deficiencies, we may classify the resulting maladies into two main groups. The first includes those caused directly by the lack of certain dietary essentials. Examples of this group are rickets, goitre, anemia, polyneuritis, scurvy, sterility, pellagra and sore mouth. The second group includes those diseases which are indirectly the result of the lowered resistance to bacterial infection, toxins and poisons caused by malnutrition. In this group may be mentioned xerophthalmia, loin disease of cattle, respira-

tory infections, sinus infections and general infectious diseases and toxemias, as typhoid, tetanus, etc.

THE IMMUNITY MECHANISM

This increased susceptibility is easily explained when we consider how dietary deficiencies affect the immunity mechanism. This mechanism consists of the following factors:

1. The intact skin and mucous membranes, which normally prevent the entrance of bacteria. We have seen that in certain deficiencies there is an atrophy of the sebaceous glands of the skin and glands of the intestinal tract. This permits the entrance of bacteria through the skin or through the mucous membranes.

2. Secretions, such as the hydrochloric acid of the stomach, which serve as antiseptics. The inhibition of these secretions by avitaminoses permits a more rapid increase in the number of certain bacteria which are normally present.

3. Glandular secretions which prevent the invasion by bacteria, by means other than that of destroying the organisms. The secretions of mucous and lachrymal glands normally keep the membranes moist and check bacterial invasion. In vitamin-A deficiency the failure of these glands to function causes a drying of the membranes and results in an accumulation of bacteria.

4. Phagocytosis. This, which is normally a very important means of defense against infection, has been shown by various authors to be much depressed in avitaminic animals.

5. Antibodies, such as opsonins, antitoxins, agglutinins, precipitins, hemolysins, bacterolysins and cytotoxins. These are all-important in normal animals in maintaining the resistance against bacteria. Our experiments show that in avitaminic animals there is a marked reduction in the formation of these antibodies. Microscopic examination of several of the components of the reticulo-endothelial system, which is associated with the production of antibodies, shows that it is markedly impaired in avitaminic animals. This impairment, in part at least, explains the diminished antibody production.

Since every factor of the immunity mechanism is impaired by a lack of the vitamins, the increased susceptibility to disease of poorly nourished animals is readily understood.

Our columnists can cast as many slurring remarks as they want to at Old Dobbin, but the fact remains that he rarely stalled on the railroad track.

STUDIES IN INFECTIOUS ENTERITIS OF SWINE*

Third Paper

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HISTORICAL

The earliest recorded literature of the disease under discussion is probably that of Roloff,¹ who, in 1862, observed and described a disease of swine characterized by a caseous and ulcerative inflammation of the intestines, especially of the mucous membrane of the large intestine, with caseation of mesenteric lymph-glands. Associated with these lesions he observed in some cases a similar affection of the lungs. He recorded the occurrence of the condition as primarily of young pigs kept in insanitary pens. This is regarded by Glasser as doubtless the same condition as the chronic paratyphoid infection later common in Germany.

Salmon and Smith, in 1885,² described an organism isolated from the spleens of sick hogs suffering from the disease then recognized as swine plague and gave it the name *Bacillus suispestifer*. This name was doubtless unfortunate, in that the disease, with which it was associated and of which it at that time was looked upon as the cause, was later recognized as an independent affection unlike the European swine plague. Salmon and Smith proposed that the name hog cholera should be given to the enteric disease common in America and sharply differentiated it from the European swine plague. Moore and de Schweinitz, in America, and Bang, Schutz, Jensen and Presiz, in Europe, confirmed the findings of Salmon and Smith.

In 1904, Dorset and de Schweinitz³ made the discovery that a disease of swine in Iowa, which clinically resembled hog cholera, was transmissible from affected to healthy animals through filtered, bacteria-free blood. Soon afterward, Dorset, Bolton and McBryde⁴ proved that this disease was identical with hog cholera and that a virus was its cause, whereas the *B. suispestifer* formerly believed to be the cause, was merely a secondary invader producing changes in the intestines of hogs already

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affected with hog cholera. The correctness of this new conception was confirmed by many prominent investigators, notably Hutyra, Ostertag, Uhlenhuth, Lechainche and Vallée and Sir Arnold Theiler.

ORGANISMS FOUND IN NECROTIC ENTERITIS

Our studies of necrotic enteritis have resulted in the isolation of *Salmonella suipestifer* from one hundred per cent of the field cases investigated. The organism isolated has been fed to more than 100 head of young swine, with practically 100 per cent production of the disease in these experimental cases. Our attempts to produce the disease by hypodermic injection of the organism into susceptible pigs have always resulted in failure. We have given attention to the part played by organisms other than the *S. suipestifer*, which have been considered by other investigators as possibly the cause of infectious enteritis. The *Balantidium coli* and the *Trichomonas suis* we have satisfactorily eliminated as specific causative agents of the disease or as important secondary invaders, since they were not present in over 50 per cent of the cases studied.

The *Actinomyces necrophorus* undoubtedly is an important secondary invader, since it was found consistently by us in all cases, at least in the later stages of the disease. In experimental cases destroyed early, before extensive caseation had occurred, the necrophorus organism was either absent or present only in limited numbers in a few short chains at the edge of the zone of caseation, numbers so limited that they could not be considered as productive of the diseased condition. The *S. suipestifer*, on the other hand, is found in such regions, often in pure culture and in great numbers. The anaerobic condition created by the injury to the tissues by the *S. suipestifer*, with accompanying exudation and necrosis of the epithelium of the mucosa and underlying tissues, favors the invasion and multiplication of the necrophorus organism.

CULTURAL CHARACTERS OF THE ORGANISM

The *Salmonella suipestifer*, a member of the colon-typhoid group, is an actively motile rod which grows vigorously in broth and on agar. It is apparently a quite definitely specialized organism of swine. Jordan⁵ states that he has never obtained this organism among strains from rodent or bovine sources, but that it may, apparently, rarely affect animals other than swine. The strains we have isolated from cases of necrotic enteritis have

produced no indol. Jordan⁶ states that, in tests of over 200 strains of the paratyphoid-enteritidis group, he has found positive indol reaction only in those that show distinct difference from the true paratyphoid types, either biochemically or by agglutination tests and the *S. suispestifer* strains studied by him have given consistently negative results.

FERMENTATION REACTIONS

Many investigations have been made to determine the fermentation reactions of the paratyphoid-enteritidis group. The colon-typhoid group, it will be recalled, is divided into three sub-groups, on the basis of acid and gas production in the three sugars—dextrose, lactose and saccharose:

Sub-group 1—Colon group produces acid and gas in all three sugars.

Sub-group 2—Intermediate or hog cholera group produces acid and gas in dextrose.

Sub-group 3—Typhoid-dysentery group produces gas in none of the sugars and acid inconsistently in dextrose.

Jordan,⁷ in a study of twenty-five organisms isolated from diseased swine, found only one strain that failed to conform to this classification. This was an organism isolated by Dorset in 1899 from the spleen of a pig dead of acute hog cholera. It resembled the typhoid organism in that it produced acid but no gas in dextrose. Ten Broeck⁸ reports a non-gas-producing hog cholera bacillus which had been carried in stock for fourteen years. In all other ways it resembled the typical hog cholera bacillus. Bainbridge⁹ also reports a strain of *S. suispestifer* which is typical in all ways except it is non-gas-producing.

Jordan calls attention to some of the discrepancies in gas production as being due to unsatisfactorily prepared carbohydrate media. According to him the three commonest sources of error rest in the use of carbohydrates not free from dextrose, in the use of meat infusion broth not free from muscle sugar, and in overheating during sterilization, leading to the formation of some dextrose. As a means of avoiding errors, two of us (McNutt and Purwin) have developed a medium highly satisfactory for use with carbohydrates in tests of the fermentation reaction of the *suispestifer* and related organisms. Its chief advantages noted are definite, clean-cut reactions, ease of manufacture with economy of production, and coagulation accompanying the formation of much acid. It consists of:

Nutrose*	1.0
Water distilled	99.0
Sodium chlorid	0.5
Andrade's indicator†	1 0

Mix the ingredients and allow to stand over night. Steam two hours in the Arnold sterilizer. Filter through cotton. Addition of carbohydrates is then made and the medium is tubed and sterilized in the Arnold for 25 minutes on three consecutive days. With this medium fermentation reactions are highly satisfactory. In a test of 172 strains of 30 different organisms, including many strains of *S. supestifer* of our own isolation as well as four European strains and eight American strains, from Jordan, Theobald Smith and Dorset, we have found remarkably clean-cut, consistent, uniform reactions.

In studies of fermentation reactions of organisms of the paratyphoid-enteritidis group on a large number of carbohydrates, there is quite general agreement by investigators regarding the reaction of the majority of these. Only three of the carbohydrates give varying results. These are arabinose, xylose and dulcite. Jordan¹⁰ found that only 4 out of 25 cultures of swine origin studied by him gave fermentation with dulcite in 24 hours, while 5 others ferment it slowly, within 15 days. These latter came from various sources in Europe and America and only one of them carried a complete history. On the other hand, he found all cultures from human sources fermenting dulcite within 24 hours.

Summing up his studies, Jordan states that the characteristic of delayed dulcite fermentation served to distinguish between the paratyphosus B and A strains, the latter in this respect approximating quite closely the *S. suipestifer* type which, however, acts negatively or not at all upon dulcite. He finds that only 4 of the 25 porcine strains, like all the A strains, ferment arabinose promptly, these being the same strains that attack dulcite, showing an exact correlation in this group of cultures. With the exception of two strains of porcine origin, Jordan found all fermenting xylose with production of both acid and gas, while they attacked arabinose and dulcite slowly or not at all. All of our strains ferment xylose but, with few exceptions, fail to act upon either arabinose or dulcite and, if at all, only after long culturing upon the media.

*Manufactured by Meister, Lucius and Bruning, Hoechst, Germany.

†Hiss and Zinsser (4th ed.; D. Appleton & Co., New York), p. 136.

OCCURRENCE IN NORMAL SWINE

Jordan,¹¹ in a study of the bacterial flora of 291 normal swine, isolated 1419 strains from the lower intestine. Only 40 of these gave any indication of being of the *suipestifer* type and of these 40 not one was agglutinated by a high-titer (5,000-10,000) *suipestifer* serum. He therefore concludes that the occurrence of true *suipestifer* strains in any abundance in the intestines of normal swine in this country is a rarity and if it does happen is to be looked upon as an expression of the "carrier" condition due to association of healthy animals with those infected.

Dorset¹⁴ reports the isolation from the intestine of a normal hog of an organism possessing the cultural characters of *B. cholerae suis* but lacking pathogenic powers for the guinea pig. Uhlenhuth and his co-workers¹² examined the intestinal contents of 600 presumably healthy swine in the Berlin abattoir. They found in 51 of these animals bacilli resembling the organism they had obtained from cholera-sick hogs. Grabert¹³ found bacilli resembling *suipestifer* in 7 out of 23 swine without intestinal lesion. Schmidt and his co-workers¹⁴ examined the feces of 700 healthy swine in the slaughter-house at Leipsig and found in about 4 per cent bacilli which culturally resembled paratyphosus B, about 1 per cent of which were agglutinated by high-titer paratyphosus B serum but few, if any, of which manifested pathogenicity for animals.

Trawinski,¹⁵ in a study of 500 healthy swine, found but two strains of organisms which, according to type of colony, biological and serological reactions and pathogenicity, were identified as *B. suipestifer*. Heinick¹⁶ isolated from the feces of 23 healthy swine 22 varieties of organisms, none of which were of the paratyphoid B. group. Aumann¹⁷ had negative results from an examination of the intestinal contents of 101 hogs, as had Soberheim,¹⁸ in the examination of 102 samples from intestines of 51 swine in Berlin. Our own efforts to demonstrate *S. suipestifer* in normal swine have been unsuccessful.

PATHOGENICITY

Of the laboratory animals, mice are probably most susceptible to artificial infection with *S. suipestifer*. Inoculation with minute quantities of bouillon culture results in septicemia and death within a few days. Rabbits and guinea pigs are quite susceptible to infection with *S. suipestifer* when fed or injected. The former offer less resistance than the latter. A series of rabbits

fed 5 cc of broth culture all succumbed within one week, showing severe diarrhea, marked enteritis in the lower bowel, swollen, hemorrhagic mesenteric lymph-glands and necrotic abscesses in all Peyer's patches and solitary lymph-nodes. Rabbits injected with 2 cc of virulent culture died from septicemia within 48 hours. Guinea pigs fed 2 cc of culture and others injected with 1 cc of culture generally died within one week. This susceptibility of rabbits made extreme care necessary in artificially immunizing them for production of agglutinins.

Our earlier experience in attempted production of agglutinins by injection of rabbits with live cultures was unsatisfactory, since before agglutinins appeared in concentration many of the rabbits died. Later the following technic was followed with success. Six injections were made at 5- to 6-day intervals. The first two injections consisted of 1 cc of killed broth culture each, given intraperitoneally. The third injection was 0.2 loopful of agar culture heated one hour; the fourth, 0.4 loopful heated one-half hour; the fifth, 0.2 loopful of live culture and the sixth, 0.4 loopful of live culture administered intravenously. Rabbits which, before this treatment, showed no agglutinins for *S. suispestifer* yielded blood with titers as high as 1 to 12,800 following treatment.

Some studies have been made by various workers to determine whether *S. suispestifer* may live in the intestinal tract of various rodents. Verder¹⁹ reports agglutination tests of the blood of 100 rats caught in Chicago packing-houses. No agglutinins for *S. suispestifer* were found. Savage and White,²⁰ in a similar investigation, found 29 rats out of 96 studied showing agglutinins for *B. enteritidis*, with 6 yielding virulent strains of the same, but none indicated infection with *S. suispestifer*. Infection in swine can be produced only by feeding virulent cultures. One strain of the organism isolated by us (No. 24) has produced typical cases of the disease when fed in doses of 20 cc of broth culture. Forty-eight-hour cultures are best. Old cultures (8 days) are toxic and produce symptoms within a few hours after feeding but the full course of the disease is not so typical as when 48-hour cultures are fed and a higher percentage of such pigs live than is the case where 48-hour cultures are used. Vomiting occurs often within a few hours, diarrhea within 24. Fever is usually manifest within 12 to 24 hours, temperatures of 106° F. occurring frequently in 24 hours.

If pigs are starved for 24 hours before feeding, the results are most satisfactory. Salmon and Smith found that neutralization of the acidity of the stomach favored infection when cultures were fed. The influence of other factors upon the pathogenicity of *S. suispestifer* is well worth consideration. In one group of pigs used by us in a feeding exposure experiment were a number of individuals badly infested with ascarids. Upon autopsy of this group it was observed that the lesions of enteritis in the worm-infested pigs were much more advanced than in the worm-free pigs given exposure at the same time. It is our thought that the changes brought about by the presence of ascarids may aid the invasion by the *S. suispestifer*, thereby affecting the course of the disease and the resulting lesions. Experiments to determine this point are now in progress.

The effect of simultaneous treatment for hog cholera administered at the same time as the *S. suispestifer* culture was noted by us in an experiment with thirteen pigs of an average weight of 40 pounds. These were given simultaneous treatment for hog cholera on the same day they were fed 200 cc each of broth culture of *S. suispestifer*. All developed necrotic enteritis and eight died within 14 days. The five others were killed. All showed good lesions of necrotic enteritis. Only two of the 13, a pig dying in 6 days and the check pig which received virus only, showed any lesions of cholera. The average period these pigs lived was 7 days. This is practically the same as the average for a large number of pigs fed culture alone and the only deduction that can be drawn is that the course of the disease caused by *S. suispestifer* in artificially infected pigs is but little, if at all, altered by serum-virus treatment for hog cholera at the same time. The lesions of cholera appearing in one of the group suggest the possibility of the simultaneous treatment for cholera being unfavorably influenced by infection with *S. suispestifer* at the same time.

In regard to the pathogenicity of *S. suispestifer* for man there is considerable variance of opinion. In a recent article⁵ by Jordan he says:

Human food poisoning due to *S. suispestifer* has occurred, but seems to be relatively rare. The ways whereby it finds its way into incriminated food seem to have remained undiscovered although pork has usually been suspected. It is remarkable that despite the frequent occurrence of hog cholera in which this organism is usually present, there are no reported instances of farm outbreaks of human food poisoning associated with outbreaks of the porcine disease.

AGGLUTININS FOR *SUIPESTIFER* IN NORMAL SWINE

Agglutinins for *S. suispestifer* in the blood of healthy swine vary. The majority of those examined show no agglutination in dilutions up to 1:20, some few as high as 1:200. The individuals in a herd of 13 pure-bred Chester White shot of about 50 pounds weight showed the following agglutinating titers:

Titer	Number
0	6
1-25	2
1-50	2
1-100	2
1-200	1

The variations manifested may have been the result of previous infection with *S. suispestifer* or of association with infected pigs, as the animals were all undersize and in unthrifty condition, though manifesting no symptoms of sickness at the time and giving negative reactions to the occult-blood test applied to the feces, a procedure which we have reported in a previous publication²¹ as reliable in the detection of pigs suffering active infection with *S. suispestifer*. Two pigs in another lot of 38 healthy pigs showed agglutinins present in a 1-200 dilution; the 36 others were negative. Wehrbein,²² in our laboratory, found that of 8 normal pigs tested 5 showed no agglutinins, 2 showed agglutinins in a dilution of 1-10 and 1 in a dilution of 1-40. In a study of 100 swine hyperimmunized for anti-hog cholera serum production, he found the average agglutinating titer to be 1-1000, ranging from 1-20 to 1-10,000. In explanation of these findings he says:

The agglutinins were produced not as a result of acute cholera but as a result of the presence of *B. suispestifer* in the large amount of virus blood injected (5 cc to each pound weight). Twenty-one of the 100 had a titer under 1-80, and in these cases I assume that no considerable quantity of *B. suispestifer* had been injected as often the blood of one virus pig was used for the hyperimmunization of one immune pig. Mixtures of different bloods were, however, the rule.

AGGLUTININS IN INFECTED SWINE

Table I records the serological reaction of swine given exposure to *S. suispestifer* in different ways. In all cases animals were bled approximately 20 days following exposure. Pigs 6 and 7 were not knowingly exposed to *S. suispestifer* but were two used in routine virus tests.

Wehrbein²² found in his study of eight virus pigs that agglutinins were absent in five cases, present in dilution 1-20 in one and

1-40 in two. He states that in his opinion more than 44.6 per cent (Uhlenhuth's estimate) of cases of hog cholera in Iowa show accompanying infection with paratyphosus B-type organisms. Eberson,²³ in the same laboratory, found 28 of 55 virus pigs carrying an infection of paratyphoid B type.

IMMUNITY TO INFECTION

The use of biologic products for the control of human typhoid and paratyphoid is quite generally considered successful. The close relationship of *S. suispestifer* to the causative organisms of these diseases encourages the investigator to a trial of similar products for controlling necrotic enteritis, especially in swine

TABLE I—Serological reactions of swine exposed to *S. suispestifer*

PIG	METHOD OF EXPOSURE	EFFECT	TITER
7948	Fed 25 cc of 24-hr. broth culture	Died	800
7947	Fed 50 cc of 24-hr. broth culture	Died	800
7945	Fed 200 cc of 48-hr. filtered broth culture	Lived	100
7944	Fed 200 cc of 192-hr. filtered broth culture	Lived	200
7939	Injected intravenously 5 cc of 48-hr. culture	Lived	800
8403		Lived	1600
7930	Fed spleen of infected pig	Killed	400
7926	Fed lung of infected pig	Killed	400
1	Pen exposure to sick pig	Lived	0
2		Lived	1600
3		Lived	50
4		Lived	25
5		Lived	100
6	Pig used in hog cholera virus test	Died	25
7		Died	0

raised on infected premises. As stated in a previous article,²⁴ there are undoubted limitations to the use of biologics in the control of enteritis that either do not exist or are more readily overcome in typhoid. Numerous investigators have shown that typhoid vaccination is not always successful where the sanitary barriers are broken down. Sanitary barriers in the human family are far more readily erected and maintained than is the case in swine-raising. The conditions under which swine live are such that, judged by human standards, there are no sanitary barriers. The habits of swine are such as to render them consumers of feces through contamination of their feeds and no practical methods of feeding have yet been developed which can prevent this. Vaccination alone has not eliminated typhoid

fever but vaccination assisted by improved sanitation and modern medical care and nursing have done so in many localities.

In considering then the efforts to control enteritis by biologic treatment, it is but fair that we should call attention to the limitations of such treatment in the field of veterinary medicine. Vaccination for this disease can never be a substitute for other means of avoiding it and in our opinion may only reasonably be expected to be auxiliary to proper sanitation. While recognizing that it has been many times demonstrated that there is no correlation of protective immunity against specific infection and the presence of agglutinins in the blood of an infected animal, nevertheless we are encouraged to hope that some protection against infection might be furnished swine by vaccination, when we observed the presence of agglutinins in the blood of pigs exposed to infection in different ways, particularly through hypodermic injection of small quantities of the organism. Certain of our experiment pigs thus treated gave evidence of marked resistance to subsequent infections.

Our first field tests with pigs that had been vaccinated or that had recovered from feeding tests were conducted on a farm where necrotic enteritis had occurred annually for several years. The last outbreak of the disease was in the early winter preceding our tests. The disease at this time was of a highly acute and virulent form. About 70 head of fattening hogs from a herd of 150 had died. Thirteen pigs of 90 pounds average weight were injected intraperitoneally with 5 cc of virulent, living culture. Ten days later (April 14) they were placed in the infected lots of the above farm associated with the spring pigs farrowed on the farm. On June 13, eleven more of 50 pounds weight similarly treated were added to the herd. At the same time nine untreated pigs were added.

On October 10, seven pigs of 70 pounds weight which had been fed virulent broth cultures on August 15, with successful infection but subsequent recovery, were added to the herd. This made a total of 31 treated and 9 untreated pigs. Of this number six died during the summer and late fall, five of them being untreated. Three of these five were obtained for autopsy and showed well-marked lesions of necrotic enteritis. The other two were never available for autopsy and it is not known whether they had the disease or not. One of the vaccinated pigs died with lesions of enteritis. Some of these hogs were kept in the breeding herd, one of them for four years. During the same period, a

number of the pigs farrowed on the farm died from necrotic enteritis.

This season we have vaccinated 200 head of pigs on four different farms, where enteritis had occurred for from one to three years or was then present. Vaccination has preceded simultaneous treatment for cholera five to seven days. The outcome has been apparently satisfactory, but one death occurring on one farm at the time of this report. These few tests are far from proving anything, as it was often impossible to leave check pigs. The only opportunity we have had to run checks has been on one farm where enteritis had occurred the two preceding years, appearing in five to seven days following simultaneous treatment for cholera with a considerable mortality. This year there was diarrhea present in a number of pigs of the herd shortly before they were ready for treatment for cholera. The herd was sorted and all pigs showing diarrhea together with all unthrifty or undersized pigs were placed in a separate lot. These (about 50) were vaccinated with 5 cc of phenol-killed culture. The remaining thrifty pigs were left untreated. In seven days all received simultaneous treatment for cholera. No pigs were lost in either lot.

A later experiment indicates that whatever immunity is acquired as the result of infection by feeding *S. suispestifer* is short-lived and in many cases low grade. July 26, 1928, thirty-seven head of 60-pound pigs were fed varying doses (50 to 200 cc) of broth culture of virulent *S. suispestifer*. Twenty-one head died or were killed. The remaining 16 were fed until October 18. They had made apparently good recoveries. On that date they were again fed massive doses of broth culture of another strain of *S. suispestifer*. All contracted the disease a second time and six of the number died. None of them appeared to be any more resistant to infection than they had been at the time of their first exposure in July and the percentage mortality was two-thirds as great. At the same time six healthy pigs that had never been exposed to infection were fed like quantities of *S. suispestifer*. All sickened and four of the number died. Their susceptibility was apparently no greater than that of the sixteen recovered pigs, although the mortality was considerably greater.

Based upon the results obtained in our limited series of tests to stimulate an active immunity to infection with the *S. suispestifer*, we are forced to the conservative conclusion that the agents

thus far utilized are of doubtful value. Further studies of this phase of the work are projected.

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DISCUSSION

DR. D. W. HURST: Dr. Murray stated that in these experiment animals that were inoculated with this organism and similarly treated, the animals that died did not show lesions of cholera. Under field conditions I have found in what sometimes is termed a break, following the simultaneous treatment, coming about five to eight or nine days after simultaneously treating pigs, petechiae of the kidneys and a highly congested and swollen condition of the lymph-glands. I wonder if that is regarded as a lesion of cholera in those pigs and whether there would be any difference between pigs inoculated experimentally with culture, or such as might pick them up in filthy lots on the farm. I think these pigs on the farm show cholera lesions, if I know what lesions are.

DR. MURRAY: I believe many of those are lesions of cholera, but there is only one way to tell, and that would be by injection of susceptible pigs with filtered blood. One pig out of the lot did contract cholera. Apparently the injection of the cholera blood simultaneously had little effect upon the course of the necrotic enteritis. On the other hand, the introduction of the organism may have had considerable to do with the effect of the vaccination.

DR. A. T. KINSLEY: I would like to inquire whether or not Dr. Murray has conducted experiments in which the cultures were fed, say one week after vaccination; in other words, at a time when the virus would be producing the greatest reaction—whether or not that would not give quite a different picture?

DR. MURRAY: I think it might very likely do that, but our test was not of that sort. I see your point. It is very possible the results would be different, but we have not tried culture feeding at the time you suggest.

A Correction

In listing the veterinary personnel of the Los Angeles County (California) Live Stock Department, in the January issue of the *JOURNAL* (p. 224), the names of Dr. L. F. Conti (San Fran. '17) and T. J. Stover (Ont. '01) were inadvertently omitted. The number of veterinarians in the department is now eighteen and all will be members of the A. V. M. A. by March 1, 1929, when several pending applications will be completed.

Thus far there has been no reply to the challenge implied in the note published in the *JOURNAL* last month.

THE ANATOMY OF THE DOG*

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In reading over this subject and knowing the time available for it on the program, it is at once clear that the whole field can not be considered, which is quite true; with this in mind I have selected a few of the more important fields, especially from their use surgically. Accidents may occur which would cause a given area to become of temporary importance which ordinarily would need no especial notice. Some parts of the digestive and genital systems will be discussed.

The effect of proper preparation of the subject and the effects produced for abdominal surgery will be pointed out on the illustrations.

Most of you, especially those who do some surgery, are aware that the omentum is the first loose structure encountered when opening the peritoneal cavity, and more of it is in the field of operation if the subject is placed in a horizontal position than if elevated slightly, which is the usual custom, when the omentum with the viscera slide anteriorly.

If the animal has been properly prepared, i. e., food withheld for twenty-four hours preceding the operation, and a dose of castor oil given the evening before, the stomach and intestinal tract will be quite empty and very little of the intestinal mass will occupy the portion of the abdominal cavity posterior to the transverse plane through the umbilicus, when the animal is elevated for operation.

If the urinary bladder has not been emptied, it may extend into the field of operation. The body of the uterus extends forward into the abdominal cavity and on the floor, lying practically on the median plane; but will vary, according to the fullness of the colon, which should be empty. When the colon is partially filled or full, the uterus is drawn to the left and the left horn will be pressed against the dorsal wall of the cavity by the colon. The cornua extend forward, diverging toward each kidney.

The ovaries lie against the posterior pole of the kidneys or near to them, which places them about midway between the last rib and the crest of the ilium.

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

The genital tract of the bitch is supplied with blood by the utero-ovarian, the uterine and the perineal arteries. The utero-ovarian, branching off the aorta, supplies the ovary, Fallopian tube and anterior portion of the cornua; the uterine, a branch of the umbilical artery, supplies the greater portion of the cornua and the body of the uterus and the vagina; the perineal artery supplies the external genital organs.

The spleen may vary in position at its ventral extremity, depending on the fullness of the stomach; with the full stomach it may come close to the median plane and about midway between the umbilicus and anterior border of the pubis.

The cecum, which varies somewhat in size, lies to the right of the median plane, its opening being nearly on the transverse plane through the umbilicus.

With the full stomach it may be crowded backward to a plane midway between the umbilicus and pubis. Its removal for the control of whip-worm infestation has proven quite successful. The cecum is supplied by the cecal branch of the ileo-ceco-colic artery which is a branch of the artery given off the anterior mesenteric; the other two branches go to the colon.

Control of hemorrhage in performing this operation, and in fact operations on any one on the viscera, is the more difficult because of the anastomoses which occur between the adjacent branches to the intestinal tract in both the arterial and venous supply.

The stomach of the dog is comparatively large and it has been estimated that for the average-size human and the average-size dog, that of the dog is three times the capacity of the human. The capacity is six to seven pints in the dog. In some of our subjects in the dissecting-room, which have been allowed to eat all they wished just before embalming, the stomach appeared to lie transversely and it extended posteriorly to a transverse plane one-third of the distance from the umbilicus to the pubis.

Convicted of Obstructing Tuberculin Test

Will Stimer, a farmer of Tompkins Township, Jackson County, Mich., was given the alternative of paying a \$100 fine or serving 30 days in jail, when he was convicted before Circuit Judge Benjamin Williams recently, on a charge of refusing to allow state veterinarians to inspect his cattle for tuberculosis two years ago. Continuance of Stimer's bond was arranged, pending an appeal to the Michigan Supreme Court.

REGULATORY WORK WITH BACILLARY WHITE DIARRHEA*

By B. T. SIMMS, *Corvallis, Oregon*

Oregon Agricultural College Experiment Station

The agglutination test for the detection of bacillary white diarrhea in poultry was applied to a commercial breeding flock of fowls in Oregon for the first time in 1915. In the ten years following, the number of such tests gradually increased. During this time, however, the Oregon State Livestock Sanitary Board did not find it necessary to give official recognition to such tests. But, in 1925, when the states of Washington, Oregon and Idaho, in the order named, issued quarantine regulations concerning this disease, a "condition, not a theory," confronted the Oregon Board. The situation was all the more important because Oregon hatcheries ship a considerable proportion of their baby chicks into Washington and Idaho. The Board had to devise a workable plan for officially bleeding and testing fowls, for the disposal of reactors, for the inspection of breeding farms and hatcheries, and for issuing certificates covering interstate shipment of breeding stock, hatching eggs, and baby chicks. This paper is an outline of the plan as adopted and followed by the Oregon State Livestock Sanitary Board.

OFFICIAL BLEEDERS AND APPROVED LABORATORIES

Since this Board does not maintain either a sufficient staff to collect the blood or a laboratory to make agglutination tests, it was necessary to appoint official bleeders and to approve official laboratories.

There are two cooperative breeder and hatchery associations in the State. Since the inspectors for these associations handle the birds owned by their members, it was economical and practicable to appoint these inspectors as official bleeders. The representatives of these two associations collected more than two-thirds of the total number of blood samples. Flocks not connected with either association were in most part bled by special appointees. In a few cases independent flocks were handled by association inspectors. Each bleeder usually was accompanied by an assistant. The flock-owners were required to have the birds con-

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

fined so they could be easily caught and to furnish sufficient help in handling the fowls. It was found that a crew of four could catch, leg-band and bleed from 60 to 100 birds per hour. One assistant caught the birds and pulled the feathers from the skin over the area to be nicked, one leg-banded the birds, and one wrote numbers on the tubes. In all instances the blood was obtained by nicking the ulnar vein with a sharp-pointed scalpel and collecting it in a 13 x 100 mm. test-tube.

The approved laboratory, to which the blood was to be sent for testing, supplied these tubes, corked, sterilized, labelled and packed in a special shipping-box. Bleeders were instructed to fill each tube from one-fourth to one-third full. As soon as the blood was collected, the tube was stoppered and placed flat on its side in the shipping-box. A sheet of paper was sometimes placed between the layers of tubes, but this was not necessary. After the box was filled a mimeographed form, giving the name and address of the owner, the number of samples collected, and the age, sex, leg-band numbers, and breed of fowls bled was filled out and placed in the box. If blood from more than one flock was included in a single box, it was necessary, of course, to fill out a form for each flock. The shipping-boxes held about 350 tubes each.

Nearly 500 flocks were bled during the 1927-28 testing season and a few more than 200,000 samples were collected. The number of samples collected in a day by a bleeder varied from a little above 1000 to less than 200, depending upon the size of the flocks, the distance the bleeder had to travel, the equipment for catching and handling the birds, and the amount and efficiency of the help furnished by the flock-owner. An average of 600 samples per day was considered good.

The cost of collecting the blood samples varied considerably. One association charged its members three cents per bird. The other association charged for its bleeders on a per diem plus mileage basis. The independent bleeders worked both on a basis of three cents per bird and per diem plus mileage. The average cost was approximately three cents per bird for taking the samples. There was an additional cost of about three-fourths of a cent per bird for the leg-bands.

The Board ruled that only those laboratories under the direct supervision of a poultry pathologist, who was experienced in testing for bacillary diarrhea, would be approved for official testing. Only two laboratories requested such approval. One

of these, the laboratory of the Department of Veterinary Medicine of the Oregon Agricultural College Experiment Station, tested 193,000 samples, or about 96 per cent of all those collected in the State.

The laboratory learned from experience that each official bleeder should have available approximately a ten-day supply of bleeding-tubes and tube-boxes, or about 5,500 tubes and 15 boxes. The boxes were numbered and certain numbers were assigned to each bleeder.

When the blood reached the laboratory the tubes were unpacked and placed consecutively in test-tube racks. If serum had already collected, the blood was kept cool until the dilutions were made. In some instances the blood was stored in a warm room to expedite the collection of serum. A laboratory assistant numbered agglutination tubes to correspond with the numbers on the tubes of blood. Then undiluted blood-serum was measured into these tubes with a capillary pipette. A well-trained worker could place serum in at the rate of five to eight tubes per minute. In using only one agglutination tube for each serum sample so that the pipette needed to be washed out following every manipulation, about 3000 to 3500 samples could be handled by one person in a day. If two tubes of antigen were used for each serum, 4,000 tubes or 2,000 samples could be handled in a day.

After the serum was placed in the tubes the antigen was added by using an all-glass multiple pipette which placed antigen in sixteen tubes at once.

It was found advisable to hold all blood samples in a cold room until after the tests had been read and recorded. When this was done, accidental damage to the antigen tubes before the test was complete did not make it necessary to collect samples again. If this was done it usually required about six days from the time a tube reached the laboratory for it to be ready to go back to the bleeder.

Tubes used for making the dilutions required about six days for each blood sample, too. If, therefore, only a single tube was being used for each test, the laboratory needed a minimum of six times as many agglutination tubes as it received blood samples in a day. If two dilutions were used the laboratory needed twelve times as many agglutination tubes. In order to have a little reserve, it was found necessary to have about thirty times as many tubes as there were blood samples received in a day.

If the single-tube test were used, enough racks to store eight times a single day's receipts of blood samples were necessary; while if the two-tube test were used, the rack capacity needed to be twelve times a single day's receipts. Incubator space was needed to store either four or eight times a day's receipts, depending upon whether the single or two-tube test was used.

All records were made in triplicate, one copy being forwarded to the flock-owner and the other to the State Livestock Sanitary Board office.

The laboratory charged a fee of seven cents per fowl for the testing but found at the end of the testing season that a little surplus had been accumulated and one cent per fowl was refunded.

DISPOSAL OF REACTORS

Flock-owners were required to send in to the State Veterinarian's office the leg-bands from the reacting fowls, accompanied by an affidavit that these reactors had been disposed of through either slaughter or sale for immediate slaughter. It was thought by some that such a system of handling the reactors might prove unsatisfactory but there was absolutely no evidence that any flock-owner failed to cooperate to the best of his knowledge and ability in disposing of these fowls.

INSPECTION OF BREEDING FARMS AND HATCHERIES

No regular system of inspection was maintained, but if any irregularity was reported or suspected, a representative of the Board made an investigation. The fact that an inspection might be made at any time and that the finding of any birds on the premises not carrying official leg-bands would be sufficient cause for refusal of the issuance of interstate certificates no doubt had a restraining influence, if there were any who might have tried to evade the ruling.

CERTIFICATES FOR INTERSTATE SHIPMENT

After flocks were tested and the State Veterinarian had evidence that the reactors had been removed, any duly authorized veterinarian in the State who had access to the test-charts was empowered to issue interstate health certificates. Nearly all of these were issued in blank form by the State Veterinarian, with the owner's name, and the owners filled these out for each consignment of breeding stock, hatching eggs, or baby chicks.

In addition to the certificates, colored labels were issued to each hatchery which had met the requirements of the Board.

The labels issued to each man bore a serial number and carried a statement to the effect that the poultry or eggs in the container bearing this label had met the requirements for interstate shipment.

The Board's rulings recognized two types of flocks, namely, "bacillary-diarrhea-free" flocks which had passed two successive annual tests with no infected birds, and "tested-and-reactors-removed" flocks from which all reactors had been removed.

In the future, different colored shipping labels will be furnished for these two types of flocks.

DISCUSSION

In many instances the hatcherymen were fairly well informed concerning the disease, but the owners of the flocks producing eggs for these hatcherymen were not. Since the official bleeders were the only representatives of the Board who came in direct contact with flock-owners, it was very important that these bleeders be prepared to answer any questions and explain the general plan of the work. When the bleeders were paid on a per-bird basis, they were inclined not to spend much time in educational work. It is suggested that they should be paid on a per diem basis and that all bleeders should be instructed to spend the necessary time in answering questions and explaining the methods of spread and control of the disease.

Much time could have been saved and needless travel avoided if there had been closer cooperation between the bleeders. Next season there will be a superintendent of bleeders who will have charge of the men in the field.

The number of blood samples reaching the laboratory each day was very variable last season. This increased the expense of labor in handling. The superintendent of bleeders will, no doubt, be able to arrange the work so that the number will be more nearly constant.

On the whole the work was done in a satisfactory manner. The amount of dissatisfaction was not any greater than is encountered in any other sanitary program at its inception.

The courts have decided the quarantines are illegal but the Oregon poultrymen are planning on continuing the testing. Indications are that there will be about half as many fowls tested this year as last. This seems proof that the poultrymen have been fairly well satisfied with the work of the State Livestock Sanitary Board.

A COMPARISON OF THE PULLORIN REACTION AND THE AGGLUTINATION TEST FOR BACILLARY WHITE DIARRHEA

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INTRODUCTION

In this paper are presented some comparisons of the results obtained at this Experiment Station with the pullorin reaction and the tube method of conducting the agglutination test for bacillary white diarrhea in the domesticated fowl. The results were collected over a period of several months and under various field conditions. We believe that the results of this study indicate certain methods of practice which will be of value to the practitioner and research worker in the field.

Due consideration was taken of the fact that, while the tube agglutination test is by no means a perfect test, it does free flocks from the disease and may be used as a basis for comparison. We are well aware of the fact that too much phenol in the antigen causes a precipitate which makes the test impossible to be read correctly, and that too little preservative allows certain phenol-fast organisms to grow in the tests, thus leading to false interpretations. The possibility of such false reactions has always been considered and great care has been exercised to avoid them in this work.

The tube agglutination test, using serum dilutions of 1 to 25, has been used as a standard. All tubes showing complete agglutination were considered as positive reactions. Those showing partial agglutination were considered as negative reactions.

Two types of pullorin have been studied: (1) Those from commercial concerns and other sources outside of this laboratory, and, (2) those prepared by us.¹

CULTURES AND ANTIGENS

The cultures for preparing antigens for the agglutination tests and our pullorin were the same in all cases. These cultures were isolated from young chicks and were typical throughout for the type. All agglutinated readily in the presence of known positive

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serum, but showed no indication of spontaneous agglutination, or agglutination in the presence of serum from non-infected birds.

The antigens for the agglutination tests were grown on standard meat-extract agar, containing Difco peptone and 0.2 per cent sodium citrate at pH 7.2. The 18-hour growth was suspended in physiological salt solution, washed twice in sterile salt solution, and stored in the ice-box in a very concentrated suspension until used. As a preservative during the storage period, 0.5 per cent phenol was added. When used for the tube test this antigen was diluted to somewhat less than tube 1 of McFarland's nephelometer with neutral physiological salt solution. The pullorins from outside sources were in part of the precipitated and dried form, and in part of cellular and other material in suspension. The ecto-pullorins prepared by us were similar to the ecto-antigens described by Bushnell and Hudson.² They were prepared by growing the cultures on agar for eighteen hours, suspending the organisms in salt solution and washing four times. The fourth washing of ten cubic centimeters for the organisms from one Kolle flask was autoclaved for 20 minutes at 15 pounds pressure. This product does not give the ordinary reaction for protein but acts as an antigen in complement-fixation tests. This product has been given the name "ectoantigen." We have used several modifications of this product but the above-described method of preparation is the simplest and gives results of equal value to the modified product.

TUBE AGGLUTINATION TEST

The tube agglutination test was accepted as standard merely because we are more familiar with it and know more of its complications, also because we have found it to be effective in freeing flocks of the disease where it has been given an adequate trial and where the usual common sense precautions of removing the reacting birds and infectious material have been observed.

We have always believed a low dilution necessary to remove all dangerous carriers and have accepted as a basis for this work the dilution suggested by the Poultry Disease Committee of the U. S. Live Stock Sanitary Association.³ This is a 1-to-25 one-tube test, incubated 24 hours at 37°C. with 0.3 per cent phenol added, or preferably the incubation should be at 45°C. without phenol. Complete agglutination in this test is considered to indicate a positive reaction. The borderline (\pm) cases with partial agglutination were considered as negative. This arbitrary

basis will probably miss a few carriers of the organism but is satisfactory for most practical purposes.

The tube agglutination test has been severely criticized and is at present considered by some as of little or no value as indicating actual carriers of the disease. When we consider only clear-cut agglutinations at dilutions of 1 to 25 as indicating actual carriers, and disregard those tests which are not clear cut, we have not encountered such wide variations in the results as have been reported from other laboratories. Most of the difficulties are due to precipitates in the presence of too much phenol, and to contamination by phenol-fast organisms. There is also greater variation in the constancy of the tests when the dilution is near the titer limit of the serum, since it is well known that agglutinins vary in amount in the serum of the same animal at different times, although they rarely disappear completely in a short period of time in infected animals. This is an added argument for the use of low dilutions.

THE PULLORIN TEST

The commercial pullorins were purchased on the open market and were of two kinds: First, a dried product, probably an alcohol precipitate of old cultures of the organism grown in broth (this was suspended in salt solution before use); second, a cell suspension in a clear liquid like salt solution.

Various methods of injecting the material were tested but the best results were obtained by introducing about 0.1 cc as nearly intracutaneously as possible. We have used the edge of the wattle, since no marked difference has been noted in the amount of reaction following the use of the edge or the side, and it is more convenient to use the edge. This amount of material causes a nodule about the size of a grain of wheat. If properly placed, there is formed a white area about 2 mm. in diameter in the epidermal layers. This spot will usually persist for several minutes. The pullorin is best injected by placing the bevel of the needle toward the outer surface. A tuberculin syringe with crutch top and a 27-gauge screw needle are necessary for this purpose.

The positive reaction consists of a persistent swelling about the size of the original made by injecting the pullorin. In many cases there are small discolored areas or nodules which persist for some time, but these are disregarded as not indicating a positive reaction. If the pullorin is put too deeply into the subcutaneous tissue there is a reaction but the edema is more diffuse and in-

volves more of the wattle.¹ In some strong reactions the entire wattle shows very marked swelling and feels hot and smooth to the touch.

COMPARISON OF RESULTS

Table I shows the comparison between the results obtained by the tube agglutination test and various kinds of pullorin. The tests were made on 3700 birds in 20 flocks. All birds were in producing flocks on farms near the College.

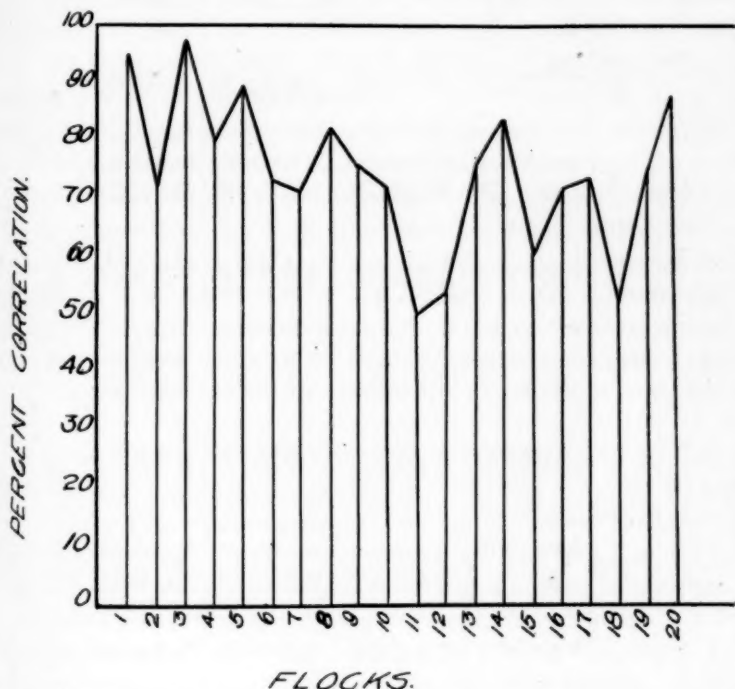


FIG. 1. Chart showing variation in the correlation between the tube agglutination and pullorin reactions in 20 flocks.

The pullorin was injected just after blood had been drawn for the agglutination test. In a few cases different pullorins were injected into opposite wattles, but this interfered with the reading of the tests and was discontinued. In two of the flocks (Nos. 3, 4 and 13, 14) the pullorin was injected twice at intervals of about two months. Both flocks contained very small numbers of reactors and the first test did not appear to affect the reaction to the second test or to increase the agglutinin content of the serum, as indicated by subsequent agglutination tests. In a small flock

TABLE I—Comparison of the results obtained by use of the pullorin and tube agglutination tests

FLOCK	BIRDS	% REACTORS AGGLUTINA- TION TEST	+A +P*		-A -P		+A -P		-A +P		CORRE- LATION	% REACTORS MISSED BY PULLORIN
			No.	%	No.	%	No.	%	No.	%		
1	185	0	0	0	177	95.7	0	0	8*	4.3	95.7	0
2	51	43.1	12	23.5	25	49.1	10	19.6	4	7.8	72.5	19.6
3	79	1.2	1	1.2	76	96.2	0	0	2	2.5	97.4	0
4	80	1.2	1	1.2	63	78.6	0	0	16	20.0	80.0	0
5	180	0.5	0	0	166	92.2	1	0.5	13	7.3	92.0	0.5
6	43	55.9	13	30.3	19	44.2	11	25.5	0	0	74.4	25.5
7	92	43.5	22	23.9	44	47.8	18	19.5	8	8.7	71.7	19.3
8	116	24.1	11	9.5	85	73.3	17	14.6	3	2.5	82.7	14.6
9	161	41.4	23	14.3	100	62.1	25	15.5	13	8.1	76.4	15.5
10	581	21.1	46	7.9	375	64.5	77	13.3	83	14.3	72.5	13.3
11	66	60.6	25	37.8	8	12.1	15	22.8	18	27.2	50.0	22.8
12	121	46.3	18	14.9	48	39.6	38	31.3	17	14.1	54.5	31.3
13	133	1.5	0	0	102	76.7	2	1.5	29	21.8	76.7	1.5
14	133	1.5	0	0	111	83.5	2	1.5	20	15.0	83.5	1.5
15	143	36.3	18	12.6	69	48.2	34	23.7	22	15.4	60.8	23.4
16	25	16.0	1	4.0	17	68.0	3	12.0	4	16.0	72.0	12.0
17	143	20.3	16	11.2	90	62.9	13	9.1	24	16.8	74.1	9.1
18	121	46.2	51	42.1	12	9.9	5	4.1	53	43.8	52.1	4.1
19	113	26.5	15	13.2	71	62.9	15	13.2	12	10.6	76.1	13.2
20	1134	8.6	8	0.7	980	86.4	89	7.9	57	5.0	87.1	7.9
	3700	17.7	281	7.8	2638	71.3	375	10.1	406	10.9	78.8	10.1

*A = Agglutination. P = Pullorin. Association coefficient 0.56.

which had been injected with pullorin several times, the treatment seems to desensitize the bird and lead to a negative reaction.

In table I no attempt has been made to differentiate individual pullorins, although there is considerable variation, as will be seen later. The discussion of the results of all types of pullorin together will be of value since similar results would be obtained under conditions of practical use in the field. The flocks used in these tests were typical of those to be met under field conditions. The percentage of reactors varied from 0 to 60.0, as based on the tube agglutination test. The association correlation has been calculated by the Yule³ formula and the percentage of correlation also has been determined. The Yule formula has not been considered except on the total group.

The percentage of correlation is irregular, as is shown by figure 1. There does not seem to be a close relation between high or low agglutinin content of the serum of the birds. Some, having high-titer serum, show no pullorin reaction, while some, without demonstrable agglutinins in the blood, show severe pullorin reactions. The exact reason for the last group of reactions is difficult to explain. It is apparently not due to tissue injury since large numbers of negative birds and some positive birds treated at the same time and in the same way show no, or very slight, reactions.

For the 3700 birds with 17.7 per cent reactors there was an association correlation of 0.66 by the Yule formula, and 78.8 per cent correlation. Such correlation does not indicate that the test is accurate enough for practical purposes. The fact that the test misses some dangerous reactors is an argument against its use.

Numerous investigators have criticized the agglutination test as not being at all accurate. All that can be said to this is that at this Station excellent results have followed its use. Similar reports come from other sources. Several flocks here have been entirely freed of all reactors as well as the disease. Such proof is the best evidence obtainable that the test is effective. On this information as a basis we have conducted this investigation.

There were 375 (10.1%) of these birds positive to the pullorin test and negative to the agglutination test; and 406 (10.9%) negative to the agglutination and positive to the pullorin test. Of these disagreements the first group is by far the more important, since they are, or have been, carriers of the organisms. Unfortunately it is not possible to distinguish between those birds which are actually and potentially dangerous carriers and birds

which have recovered and are immune. According to the above discussion we can probably say that the pullorin test is effective in detecting all but about 10 per cent of the actual carriers. Such a condition is not a safe one in disease control. It has been reported on numerous occasions that a very small number of carriers have caused heavy losses. For this reason the only safe procedure is to remove all reactors from the flock.

An attempt has been made to determine the cause of the irregularity between the two tests. Considering that the aggluti-

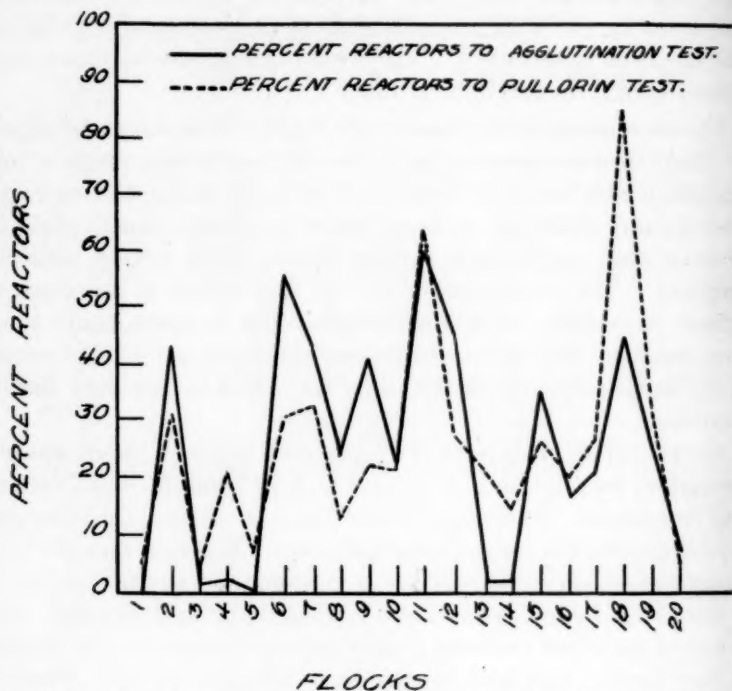


FIG. 2. Chart showing the relation between reactions to the agglutination and the pullorin tests in 20 flocks.

nation test indicates only true antigen-antibody reactions, the pullorin test should parallel it if the positive pullorin reaction is of a similar nature. An examination of figure 2 will indicate that there is somewhat of a parallel relation between the reactions and that it is probable that a properly prepared and applied pullorin will indicate very closely those birds which have reacted to the action of a homologous antigen. It is evident that the pullorins used in these tests and the conditions under which they are used in the field will not obtain accurate information of the carrier

condition in flocks under field conditions. There is no reason to believe, however, that such a product cannot be prepared.

CONCLUSIONS

While figure 2 shows some relation between the two tests on the same flock, figure 1 shows that there is not a close correlation between the reactions in different flocks.

We must conclude from this study that the pullorin test in its present status is not so satisfactory in detecting carriers of bacillary white diarrhea as is the agglutination test. Since there is a fairly high correlation between the two reactions in the same flock, it is evident that there is a possibility of developing a pullorin which will be satisfactory for the purpose. Until such a product is developed, the agglutination test should be used. The rapid, slide-agglutination test is as effective as the tube test and may be used to replace it.

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Control of Sheep Tick Described

Though widely prevalent, especially among close-herded range flocks, the blood-sucking parasite known as the sheep tick can be readily eradicated. Farmers' Bulletin 798-F, "The Sheep Tick and Its Eradication by Dipping," just revised, describes and illustrates this enemy of the sheep industry.

"The only practical way of destroying the pest," declares Dr. Marion Imes, the author of the bulletin, "is by dipping the sheep. Two dippings are necessary about 24 days apart, as the first dipping may not destroy all the eggs and these may subsequently hatch a new brood." Several kinds of dips are used successfully, the selection being largely a matter of availability, economy and convenience.

Many farm flocks of the United States as well as those kept under range conditions harbor sheep ticks and in some cases the parasites are present in sufficient numbers to cause considerable damage. The bulletin describes and illustrates the parasites in different stages of growth, gives full directions for dipping, and includes plans of wooden and concrete vats suitable for both small and large flocks.

SOME OBSERVATIONS ON THE INNERVATION OF THE UDDER AND TEATS OF THE COW AND THEIR RELATION TO MILK-PRODUCTION

By M. A. EMMERSON, Ames, Iowa

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During the course of a three-year study of the macroscopic anatomy of the bovine udder and teats,¹ several attempts were made to find a successful means of blocking the nerve supply to the udder with a local anesthetic, in order that operations might be performed upon that organ. All of these attempts were unsuccessful, as far as eliminating the sensory nerves to the udder was concerned, but observations were made that might be of interest to physiologists and those engaged in the study of the phenomena of milk-secretion.

Several grade milk cows were available for this study. The first attempt to block the nerves to the udder was made on a grade Holstein cow. A 2 per cent solution of apothesine was prepared. Approximately 5 cc of this solution was injected around the inguinal nerves of both sides, just at the point where they emerge from the external inguinal ring. This injection was made with a six-inch 22-gauge needle, designed by Dr. H. E. Bemis for use in dental surgery of the horse. The needle was inserted from the posterior basal border of the udder toward the external inguinal ring.

Following the injection it was necessary to wait twenty minutes before results were obtained. It was noticed at the end of this time that milk began to drop from the ends of the teats. The animal had been milked but a very few hours prior to this injection. Milk continued to drip from all teats for a period of forty-five minutes. Attempts to operate on the teats following the blocking of the inguinal nerves were extremely unsuccessful and met with considerable resistance on the part of the animal.

Following the above experiment, another animal was chosen and her mean daily milk-production was obtained over a period of ten days. This proved to be 21.49 pounds per day. The animal was milked as usual in the morning and evening of the eleventh day and milk weights recorded. Following the evening milking, the inguinal nerves of both sides were anesthetized with

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5 cc of a 2 per cent solution of apothesine. Twenty minutes later, she was milked again and produced 3.7 pounds of milk, making the total for that day 23.9 pounds. The following day her production totalled 22.9 pounds, or one pound less. For the nine-day period following the injection of the local anesthetic, this cow produced a daily mean production of 24.2 pounds of milk.

It might be stated here that prior to the anesthetizing of the inguinal nerves to the udder, the cow was milked three times daily, while following the experiment she was milked twice daily. Just what factor was responsible for the increase in daily production, following the anesthetizing of the inguinal nerves supplying the udder, is not known. There was no change in caretaker, feed or surroundings throughout the 20-day period. However, the interesting feature is that this animal gave 3.7 pounds of milk following the blocking of the inguinal nerves after she had been milked dry at the evening milking.

The third experiment was carried out on a grade Guernsey cow. This animal was milked morning, noon and night, for five days, to obtain her mean daily milk-yield of 22.7 pounds. On the sixth day, the inguinal nerves on each side were blocked with 5 cc of a 2 per cent apothesine solution, just twenty minutes before the noon milking. The same procedure was followed on the seventh day. The mean daily milk-production for the sixth and seventh days was 22.3 pounds. The interesting feature regarding this experiment was that the morning milkings were normal, the noon milkings were greater than any of the previous noon milkings, and the evening milkings were greatly decreased. The average mean daily milk-yield for three days following the experiment was 22.1 pounds.

The fourth experiment was performed on a pure-bred Jersey cow, 7½ years of age. This animal injured herself on a tie-chain. An accurate diagnosis was not made during the animal's life but upon postmortem it was found that she was suffering from a traumatic gonitis and that the lateral patellar ligaments were ruptured at their tibial attachments. The hip and croup muscles atrophied from disuse. This animal gave birth to a normal calf, January 26, 1927. On May 4, 1927, she was placed on experiment. One half of the udder was milked at a time and the milk weighed each morning and night for ten days. After the evening milking of the tenth day, the animal was placed on the operating-table and the inguinal nerves supplying the glands

on the right side only were sectioned, just at their emergence from the inguinal canal. Following the operation, the sphincters of the right teats relaxed and allowed the escape of milk. Milk was lost more or less continuously from the right half of the udder during the night. The right half milked much more easily the next morning than it had previously, and by the following evening the teat sphincters of the operated side had recovered sufficient tone to retain the milk in a normal manner.

Tables I and II show the amounts of milk given by each half of the udder during the entire experiment.

TABLE I.—Normal milkings of ten-day period prior to operation (Gilchrist cow).

	MAY									
	4	5	6	7	8	9	10	11	12	13
Left half of udder (morning).....	6.5	6.0	6.2	4.0	6.6	6.3	6.3	6.9		9.6
Right half of udder (morning).....	10.6	10.0	10.6	9.5	9.4	10.2	9.2	10.5		10.4
Left half of udder (evening).....	4.8	4.6	4.2	4.0	4.3	4.0	4.3		4.6	4.2
Right half of udder (evening).....	6.5	6.3	5.6	5.5	6.2	6.4	5.4		5.4	6.0

Amounts expressed in pounds.

TABLE II.—Milkings after operation (Gilchrist cow)

	MAY									
	14	15	16	17	18	19	20	21	22	23
Left half of udder (morning).....	4.8	6.2	6.1	6.0	6.4	8.3	7.6	5.6	6.1	6.4
Right half of udder (morning).....	9.2	9.3	9.4	8.0	8.3	9.7	10.1	8.5	8.0	9.3
Left half of udder (evening).....	4.2	4.4	4.2	5.1	4.2	4.0	4.2	4.1	4.0	4.2
Right half of udder (evening).....	5.0	5.8	5.1	7.2	5.5	5.3	5.1	4.7	5.7	5.1

The mean amount of milk secreted by the left half of the udder for the ten-day period prior to the operation was 5.42 pounds per milking, while that of the right half was 7.97 pounds per milking. Following the operation, the left half secreted a mean amount of 5.28 pounds per milking, while the right (denervated) half secreted a mean of 7.21 pounds per milking. The difference in daily production of the entire organ before and after operation was only 0.9 pounds, in favor of the post-operative yield.

CONCLUSIONS

1. Montane and Bourdelle² state that the sympathetic nerves to the udder of the cow are incorporated in the inguinal nerve-trunks. The first three experiments would tend to substantiate their statement. It is known that the udder and teats contain smooth or involuntary muscle and that such muscle is supplied by sympathetic fibers.

2. The results of the fourth experiment would tend to support the belief that the nervous system plays a minor role in the direct control of milk-secretion and that secretion of milk is practically normal when the innervation is destroyed and the muscular tissue readjusts itself. This is essentially in harmony with what most physiologists hold regarding the direct influence of the nervous system on milk secretion.

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DISEASE CONTROL IMPORTANT IN RABBIT RAISING

Success in raising rabbits depends to a great extent on the ability to keep the animals free from parasites and bacterial diseases. A knowledge of the more serious ailments is advantageous, says the United States Department of Agriculture, and may be had from a study of Farmers' Bulletin No. 1568-F, "Rabbit Parasites and Diseases," a new publication now ready for distribution to persons requesting it.

Among the parasitic diseases given special attention in the bulletin are coccidiosis, ear mange or ear canker, skin mange, and stomach-worm diseases. Other parasitic affections, such as irritations caused by fleas, lice, and intestinal worms of various sorts, while not commonly producing very marked symptoms in infested animals, may gradually render them weak and unthrifty, making them more susceptible to other diseases as a result of their lowered vitality.

Prevention and control measures are more practical than treatment, and are usually matters of sanitation. In the case of parasitic diseases such measures must be based on a knowledge of the habits and life histories of the parasites involved and on their mode of transmission from one animal to another. The bulletin also discusses bacterial and other diseases, such as snuffles and pneumonia.

NITROGEN EXCRETION OF HOGS ON VELVET-BEAN RATIONS

By EMERSON R. MILLER, Auburn, Ala.

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Waterman and Jones¹ have found that dialyzed velvet-bean protein is only 32.3 per cent digestible *in vitro* as compared with 61.4 per cent for casein. Finks and Johns² reported that there was lack of growth when albino rats were fed a ration containing dialyzed velvet-bean protein, either with or without the addition of cystine. B. Sure³ considers the nutritive inadequacy of velvet-bean protein to be due to its indigestibility and amino-acid deficiency.

These reports all agree in assigning to velvet-bean protein a low coefficient of digestibility, whether it is or is not biologically correct. Aside from this, however, there is another factor which has an influence on the nutritive value of the velvet bean.

There has been identified in the velvet bean an amino-acid⁴ which, according to Gugenheim,⁵ causes, in man, nausea and vomiting, and we know from our own experience that the eating of one or two ounces of velvet beans caused nausea, headache and a feeling of heaviness and distress in the stomach, which persisted for quite a while.

In addition to this amino-acid we have also separated a nitrogenous base which has a bitterish, somewhat acrid and disagreeable taste, with indications that it, also, produces headache and digestive disturbance. Whether the latter substance exists as such in the velvet bean or is formed during the process of extraction is not yet known.

Undoubtedly, the presence of such substances in a food or feed-stuff would detract more or less from the nutritive value, even if the protein were, normally, easily digested and biologically sufficient.

In our experiments we observed in a number of cases, that when hogs on a velvet-bean ration were slaughtered, six to eight hours or longer after feeding, the stomachs were generally filled with a more or less solid mass of velvet beans which, though sour smelling, had, apparently, undergone little change. In two cases the contents of the stomach were weighed. Hog 1, weighing 265

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pounds when slaughtered, had consumed a daily average of 3.77 pounds of velvet beans as the sole ration. The contents of the stomach weighed 3150 grams, or nearly seven pounds. Hog 2 weighed 255 pounds when slaughtered and had consumed a daily average of 3.43 pounds of velvet beans and corn, half and half. The contents of the stomach weighed 1960 grams, or 4.3 pounds. In the latter case the injurious substances contained in the velvet bean had been reduced 50 per cent in the ration and consequently digestion had proceeded more nearly normally.

Aside from the individuality factor, it might be expected that if equal amounts of protein were ingested, the nitrogen content of the feces would express approximately the relative digestibility of the protein of the different rations. With this thought in mind we have estimated the amount of nitrogen in the feces of a number of hogs, some on velvet-bean rations and some on other rations.

The data recorded in table I were obtained from feces of five hogs on a velvet-bean ration and four on other rations. It will be noticed that with one exception the percentage of nitrogen in the velvet-bean feces is considerably higher than that from other rations.

In every case the hogs on a velvet-bean ration ate less feed than was eaten by the hogs on the other rations. Calculations based on the amount of nitrogen contained in the different rations and the amount of ration eaten show that the velvet-bean hogs actually consumed less protein than was used by hogs on the other rations. Notwithstanding this fact, the nitrogen content of feces from the velvet-bean hogs is in some cases more than twice as high as that of feces from other rations.

Although this does not give a definite measure of the digestibility of velvet-bean protein, it does show that its digestibility is much less than the protein of the more common rations, though this may be due, in part, to the presence of the objectionable substances already mentioned.

All results shown in the table were calculated on the dry basis.

Velvet beans contain probably not less than four or five per cent of 3-4-dihydroxyphenyl alanine, a non-assimilable amino-acid, but it has little to do with the high nitrogen content of velvet-bean feces.

A small amount of this amino-acid is probably oxidized in the alkaline contents of the small intestine and the product unabsorbed, as is indicated by the dark color of the feces. On the

TABLE I—Showing the composition of feces from hogs on velvet-bean and other rations

Ration	1	2	3	4	5	6	7	8	9	10	11	12
	Velvet Beans Razor-back	Velvet Beans	Velvet Beans	Velvet Beans Hog I	Velvet Beans Hog IV	Velvet Beans Hog III	Velvet Beans Hog III	Velvet Beans Hog II	Whole Peanuts	Corn Peanut Meal	Corn Tankage	Corn 2, High Grade Peanut Meal 1
Moisture	60.0	(5.70)	(6.18)	6.14	(5.74)	(6.77)	5.90	(4.13)	3.00	3.97	4.13	4.96
Ash	3.05	7.54	6.60	6.32	5.00	4.59	5.72	8.80	9.37	6.46	15.76	7.36
Nitrogen				6.55	6.13	7.23	7.80	5.46	2.71	3.85	4.85	4.31
P ₂ O ₅				1.97			1.89		2.47	1.82	3.66	2.44
CaO				0.80			0.78		1.54	0.70	3.23	0.42
Fiber				8.61			9.41		27.73	26.34	9.63	18.88

other hand there is abundant evidence that it is largely and readily absorbed and eliminated as such in the urine.

A section about one foot long was removed every six feet from the small intestine of hog 1. In two sections a small, rather firm lump of dark-colored material was found, otherwise the contents were semifluid and in rather small amount. The contents of the first seven sections gave a slight reaction for the amino-acid, but sections eight and nine gave negative results.

In a similar manner the contents of the large intestine were examined but no evidence was obtained of the presence of the free amino-acid. Several samples of feces also were examined but negative results were obtained.

In experiments with human subjects it was found that urine voided fifteen minutes after eating ten grams of cooked velvet beans gave a distinct reaction for the amino-acid. Urine voided three and one-half hours later did not give the characteristic tests.

PRESENCE OF INDOL

Qualitative tests showed the presence of considerable amounts of indol in the feces from hogs on a velvet-bean ration, but comparatively little in the feces of hogs on other rations used in this experiment. This would very probably show that velvet-bean protein contains relatively much of the amino-acid tryptophane and that the protein of the other rations contains very little.

This might raise the very interesting question whether or not there is any correlation between the tryptophane content of a protein and its coefficient of digestibility.

There are indications that a considerable amount of valeric or isovaleric acid is contained in feces from hogs on a velvet-bean ration and very little, if any, in the feces from hogs on other rations. To indol and this acid are to be attributed, no doubt, the very disagreeable, characteristic odor of pens in which hogs are kept on a velvet-bean ration.

URINE FROM HOGS ON VELVET-BEAN RATIONS

The urine from hogs on a velvet-bean ration turns brownish red soon after it is voided and finally becomes black. Its specific gravity, percentage of solids and percentage of total nitrogen are much higher than they are in urine from hogs on ordinary rations.

The following data were obtained for two samples of urine from a hog fed exclusively on velvet beans.

Total nitrogen per 100 cc:

- (1) 1.42 grams
- (2) 1.47 grams

Solid matter per 100 cc:

- (1) 10.09 grams
- (2) 10.42 grams

For a sample of urine from a hog on a ration of peas, potatoes and sour milk, Dr. N. G. Covington reported data as follows: total nitrogen per 100 cc, 0.608 grams; dry substance, 2.768 grams per 100 cc.

CONCLUSIONS

1. Hogs assimilate a much smaller amount of the nitrogen of the velvet bean than is utilized from other ordinary rations.
2. A relatively large amount of the nitrogen of the velvet bean is eliminated in the urine.
3. Feces from hogs on a velvet-bean ration contain considerably more indol than is usually present when other common rations are used.

ACKNOWLEDGMENT

Credit is due Dr. I. S. McAdory, of the Veterinary College, under whose supervision the slaughtering of hogs was done.

REFERENCES

- ¹Waterman and Jones: *Jour. of Biol. Chem.*, xlvii (1921), p. 285.
- ²Finks and Johns: *Amer. Jour. Phys.*, lvii (1921), p. 61.
- ³Sure, B.: *Jour. Metab. Res.*, iii, p. 373.
- ⁴.....: *Jour. of Biol. Chem.*, xlv, (1920), p. 481.
- ⁵Gugenheim: *Zeit. Phys. Chem.*, lxxxviii, (1913), p. 276.

Promotions Announced at Parke, Davis & Co.

According to an announcement recently made by Mr. Oscar W. Smith, president of Parke, Davis & Co., Dr. A. William Lescohier has been appointed general manager of the company. Dr. Lescohier has been connected with Parke, Davis & Company ever since his graduation from the Detroit College of Medicine in 1909. In 1918 he was placed in charge of the manufacture of biological products and in 1925 he was appointed director of the Department of Experimental Medicine.

Dr. Louis Klein, formerly manager of the Department of Medical Service, has been appointed promotion manager, and Mr. Ralph G. Sickles has been promoted to the post of advertising manager.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

A CASE OF PERIODIC OPHTHALMIA TREATED WITH MERCURIC IODID

By R. L. HECTORNE, *Urbana, Ill.*

*Laboratory of Animal Pathology and Hygiene,
University of Illinois*

Several months ago, Kelser¹ reported the possible value of mercuric iodid in the treatment of periodic ophthalmia in horses by the intravenous injection of red iodid of mercury. This report attracted our attention for the reason that a chronic case of this disease was under our observation. This animal, a pure-bred Percheron mare, had suffered periodic attacks of ophthalmia for several months. Notwithstanding that the vision had been seriously impaired, it was decided to administer red iodid of mercury intravenously.

The treatment began August 24, at which time the animal was almost blind. Intravenous injections in doses of 0.5 gram of red iodid of mercury, suspended in 30 cc of sterile distilled water, were administered on the following dates:

8-24-28	9-19-28	10-5-28
8-27-28	9-21-28	10-8-28
8-29-28	9-23-28	10-10-28
9-6-28	9-25-28	10-12-28
9-8-28	9-27-28	10-14-28
9-11-28	9-29-28	10-16-28
9-13-28	10-1-28	10-18-28
9-15-28	10-3-28	10-26-28
9-17-28		

In preparing the red iodid of mercury for injection it was carefully weighed and mixed with sterile distilled water. A mechanical shaker was employed to distribute the particles as evenly as possible. The mixture was then poured into a glass syringe aseptically. Care was exercised to avoid injecting the drug into any of the surrounding tissues. During and following

the treatment, observations of the animal were made. As far as could be determined, no change was observed in the vision in the way of impairment or relapses. On January 1, 1929, the vision of the animal remained impaired and it is evident that the attacks of periodic ophthalmia had so altered the structures of the eye that treatment was of no avail.

REFERENCE

¹Kelser, R. A. The treatment of periodic ophthalmia with mercuric iodid. *Jour. A. V. M. A.*, lxxiii (1928), n. s. 26 (1) pp. 93-94.

Foot-and-Mouth Disease Appears in California Again

An outbreak of foot-and-mouth disease in a herd of garbage-fed hogs on a ranch near Whittier, Los Angeles County, California, was reported by Dr. J. R. Mohler, Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, on January 18, 1929. The following day the entire herd of 3500 hogs was slaughtered and buried in the hope of stopping the outbreak at its origin. There were no indications that the infection had spread from the one ranch on which the disease was detected. Federal and state quarantines were issued immediately.

The source of the outbreak, though still under investigation, appears to have been infected garbage from a ship that had visited a foreign port and later docked at San Pedro, California. A positive diagnosis of foot-and-mouth disease was made by Dr. Rudolph Snyder, of the Federal Bureau of Animal Industry, Dr. Jacob Traum, of the University of California, Dr. J. P. Iverson, state veterinarian of California, and Dr. L. M. Hurt, of the Los Angeles County Live Stock Department.

The infected herd was located on premises well isolated from other live stock and the outlook for the prompt eradication of the infection is unusually favorable. As a safeguard to the live stock industry, however, a quarantine zone has been established and a corps of veterinarians experienced in fighting foot-and-mouth disease was on the scene of action within twenty-four hours following the establishment of a positive diagnosis.

The Associated Press recently announced that the League of Nations had sent a questionnaire to all nations, concerning the importation, transportation and exportation of animals for the purpose of coordinating the veterinary health service in all parts of the world. If the nations approve, the League will publish veterinary bulletins regularly, according to the announcement.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

So much of War Department orders as assigned Captain Charles S. Williams to Camp Knox, Kentucky, is amended so as to assign him to Fort Reno, Okla., upon completion of his present tour of foreign service.

Reserve Corps

New Acceptances

Karstendick, Edward Francis.	Capt.	1233 Dryades St., New Orleans, La.
Morse, John Robertson.	Capt.	E. Main Extension, Middletown, N. Y.
Patton, John Wesley.	1st Lt.	432 Butterfield Drive, East Lansing, Mich.

Separations

Staley, Raymon M.	Lt. Col.	Appointment terminated 11-27-28.
Fawver, J. Roy.	Capt.	Failed to accept reappointment.
Fretz, Victor C.	2nd Lt.	Failed to accept reappointment.
Stafseth, H. J.	1st Lt.	Failed to accept reappointment.



ON THE EQUATOR

Lieutenant Colonel Robert J. Foster, Veterinary Corps, U. S. Army, and Mrs. Foster, standing on the equator, fifteen miles north of Quito, Ecuador, November 2, 1928. The equatorian line passes through the notch in the mountains at the right of Col. Foster.

AMERICAN VETERINARY MEDICAL ASSOCIATION
Minutes of Special Meeting of the Executive Board of the
A. V. M. A.

A special meeting of the Executive Board of the A. V. M. A. was held at the Hotel La Salle, Chicago, Ill., Tuesday evening, December 4, 1928, at 8:00 p. m.

The following were in attendance:

Members of the Board:

Dr. George Hilton, District No. 1.
Dr. E. P. Althouse, District No. 2.
Dr. L. A. Merillat, District No. 3.
Dr. C. H. Stange, District No. 5, Chairman
Dr. R. S. MacKellar, Member-at-Large
Dr. T. E. Munce, President
Dr. M. Jacob, Treasurer
Dr. H. Preston Hoskins, Secretary-Editor
Drs. N. S. Mayo, C. A. Cary and T. A. Sigler, as members of the
Special Committee on Permanent Home.

The meeting was called to order by Dr. Stange and the first business was a report from the Special Committee on Permanent Home. In the absence of any formal report from the Committee as a whole, individual members of the Committee expressed their views on various aspects of the proposition. Dr. Merillat and Dr. Mayo spoke at considerable length concerning different locations in Chicago. Dr. Merillat moved that the Committee be continued and that the Secretary-Editor be made a member of the Committee. Motion was seconded by Dr. Hilton and carried.

The next order of business was the fixing of the time and duration of the 1929 meeting. The Secretary reported the situation in Detroit, with reference to other conventions, hotel accommodations and so forth. It was moved by Dr. MacKellar that the 1929 convention be held in Detroit on August 13-14-15-16. Motion was seconded by Hilton and was unanimously carried.

Dr. MacKellar next reported for the Committee on Humane Society Hospitals. He presented a prepared report, including a proposed code of ethics that had been drawn up jointly by the A. V. M. A. Committee on Humane Society Hospitals and a similar committee from the American Humane Association. Dr. MacKellar moved that the Board approve the report and recommend it to the American Veterinary Medical Association for adoption. Motion was seconded by Dr. Hilton and unanimously carried.

The Secretary then read a number of invitations received from various organizations, asking the A. V. M. A. to send representatives to meetings of these bodies. These were ordered laid on the table, by motion duly made, seconded and carried.

The Secretary then read various communications on different subjects for the information of members of the Board. No specific action was taken on any of these.

Consideration was then given to the present status of the proposed amendment to have a Section on Education. It was agreed to leave the matter in the hands of the Committee on Program until the meeting in Detroit, in 1929.

The Secretary asked for suggestions, by which it might be possible to make the A. V. M. A. of more value and interest to the seven student chapters of the A. V. M. A. which have already been organized in as many different veterinary colleges. Numerous suggestions were made by different members of the Board.

The Board approved a recommendation of the Secretary that the proceedings of the 1928 meeting of the United States Live Stock Sanitary Association be published in the March, 1929, issue of the JOURNAL of the A. V. M. A., on terms to be agreed upon jointly by the secretaries of the two associations.

The Secretary submitted a proposal of the National Electric Sign Works for supplying members of the A. V. M. A. with a standard electric sign, showing the emblem of the A. V. M. A. The proposal was approved by the Board and the Secretary was instructed to work out the details of the arrangement.

Dr. Merillat directed attention to the fact that he planned to attend the meeting of the Central Committee of the International Veterinary Congress, to be held in Paris in May. It was moved by Dr. Hilton and seconded by Dr. Althouse that Dr. Merillat be designated as the official spokesman of the A. V. M. A. at this meeting, but at no expense to the Association. The motion was unanimously carried.

It was moved by Dr. Hilton and seconded by Dr. Merillat that the expenses incurred by Drs. MacKellar and Althouse in attending this meeting of the Executive Board be paid by the Association. The motion was unanimously carried.

There being no further business to come before the Board, a motion to adjourn was seconded and carried.

H. PRESTON HOSKINS,
Secretary-Editor.

ASSOCIATION MEETINGS

CONNECTICUT VETERINARY MEDICAL ASSOCIATION

The autumn meeting of the Connecticut Veterinary Medical Association was held in Danbury, Conn., November 7, 1928.

A large-animal clinic was held at Dr. V. M. Knapp's hospital, under the supervision of Dr. J. N. Frost, of Ithaca, N. Y. Dr. Frost performed several major operations and discussed many subjects before one of the largest meetings of this Association.

In addition to the regular members, about ten visitors were present from other states.

E. H. PATCHEN, *Secretary.*

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-first annual meeting of the Nebraska State Veterinary Medical Association was held in Omaha, December 11-12-13, 1928. Headquarters were at the Rome Hotel. Dr. W. T. Spencer, of Lincoln, presided.

Following the opening ceremonies, several very interesting papers were presented, following which Dr. A. T. Kinsley, of Kansas City, Mo., presided over a round-table discussion on hog diseases. The evening was given over to the annual banquet, followed by dancing.

The program the second day was opened with a short business session followed by papers and addresses. Officers for the ensuing year were elected as follows: President, Dr. E. C. Jones, Grand Island; vice-president, Dr. H. L. Feistner, Auburn; secretary-treasurer, Dr. Bernhard Witt, Scribner; members of the Executive Board, two-year term, Dr. J. W. McGinnis, Ord, and Dr. D. W. Hurst, Tecumseh; one-year term, Dr. H. A. Simon, Coleridge, and Dr. F. Perrin, Lincoln.

A new Constitution and By-laws was adopted. Under the same, the retiring president automatically becomes chairman of the Executive Board for the ensuing year. Grand Island was selected as the meeting place for 1929. A very encouraging sign was the election of thirty-two new members, the largest number to join the Association at any one time.

On the third day a clinic was held at the Sales Pavilion of the Union Stock Yards Co., in South Omaha. The program included

demonstrations of the use of the emasculatome and the equine stomach-tube; examinations for lameness; methods of restraint; surgical operations on swine and cattle.

F. PERRIN, A. V. M. A. *Res. Sec. for Nebraska.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Academy of Medicine Building, 103rd St. and Fifth Ave., Wednesday evening, January 2, 1929. President H. K. Miller presided.

The minutes of the December meeting were read and approved with the exception of that part which dealt with Dr. Jackson's application, same being stricken from the records. The President read a paper on "Events of Veterinary Medicine During the Past Decade," which also included several recommendations pertaining to changes in the Constitution.

The committees for 1929 were announced as follows:

Program: Dr. C. G. Rohrer, Dr. R. S. MacKellar and Dr. J. E. Crawford, Sr.

Membership: Dr. Alexander Slawson, Dr. James Harding, Dr. Jack Crawford and Dr. Alfred Meyer.

Legislation: Dr. Ray W. Gannett, Dr. Charles S. Chase and Dr. Edwin R. Blamey.

Prosecutions: Dr. O. E. McKim, Dr. L. W. Goodman, Dr. E. A. Durner, Dr. Herman Kock and Dr. J. Lebish.

Humane Societies: Dr. R. S. MacKellar, Dr. Ray W. Gannett, Dr. Edwin R. Blamey and Dr. Bruce Blair.

Dr. J. E. Crawford read an interesting and instructive paper on a modern dog hospital, management, hospitalization of patients and personnel. Some of the salient points were: (1) location, (2) pleasing architectural construction, (3) necessary arrangements for sanitation and drains, (4) keeping of records and books and (5) sending bills out promptly and demanding a retainer in all cases. Dr. Crawford summed up his talk by saying that if service were given money would come automatically. Dr. Woodward spoke on interesting hospitals and management of same. Dr. Goubeaud spoke on the rabies situation in Queens. Dr. Slawson spoke on his experiences with rabies.

Dr. Chase reported a case where the State Board of Health had been inconsistent in sending the laboratory report of a sus-

picious case of rabies direct to the owner and the health officer and had ignored the veterinarian.

Dr. R. S. Mac Kellar read the tentative agreement as passed by the American Humane Association in regard to humane hospitals and veterinarians.

Dr. Alexander Slawson stated that he believed the individual doctor should use radio publicity as do the medical doctors.

Dr. E. R. Cusaing, of Plainfield, N. J., stated that the meeting of the New Jersey State Veterinary Medical Association would be held at the Plaza Hotel, Journal Square, Jersey City, on January 31, 1929.

RAYMOND J. GARBUTT, *Secretary.*

CALIFORNIA VETERINARY CONFERENCE

The annual Veterinary Conference given jointly by the California State Veterinary Medical Association and the Division of Veterinary Science, University of California, was held at University Farm, Davis, January 2-5, 1929.

The program contained the names of two prominent veterinarians from outside the State: Dr. Hadleigh Marsh, of Montana, and Dr. J. V. Lacroix, of Illinois. Those present were greatly pleased to learn that Dr. Maurice C. Hall, Chief of the Zoological Division, U. S. B. A. I., Washington, D. C., would be one of the speakers at the Conference, although his name did not appear on the program, as Dr. Hall had only recently decided to make the trip to California.

Dr. Hadleigh Marsh, of Helena, Montana, delivered a series of four lectures on subjects relating principally to diseases of sheep. His first lecture was entitled, "Infectious Diseases of Sheep." The subject of his second lecture was "Anaerobic Infections of Sheep." In his third lecture he spoke on "Plant Poisoning In Domestic Animals," while in his last lecture he discussed "General Diseases and Parasites of Sheep."

Dr. J. V. Lacroix, of Evanston, Illinois, gave six lectures on subjects of interest to small-animal practitioners, as follows:

- "Small-Animal Hospitalization and Hospital Management."
- "Intestinal Worm Parasites of Dogs."
- "Canine Distemper."
- "Fractures and their Treatment."
- "Intestinal Derangements of Dogs."
- "Diseases of the Skin of Dogs."

Dr. Maurice C. Hall, of Washington, D. C., gave two lectures. His first lecture was on parasitology in general. He also gave in detail the operation of the Zoological Division of the United States Bureau of Animal Industry. Dr. Hall's second lecture was on "Intestinal Parasites of Dogs," which was followed by a question-box, dealing with parasites in general.

Dr. G. H. Hart and staff showed the veterinarians through the new Animal Science Building. This building, which was recently completed at a cost of nearly \$300,000.00, is a decided addition to the buildings on the college grounds.

Dr. Karl F. Meyer, Professor of Bacteriology, University of California, at Berkeley, gave a highly instructive and interesting lecture, the subject being "Some Intercommunicable Diseases of Man and Animals." Dr. Meyer also gave an illustrated travelogue. The pictures shown and described were largely mountain scenes of Switzerland, together with numerous views taken in national parks in the United States.

Dr. Agnes Fay Morgan, connected with the University of California, at Berkeley, described in detail certain experiments that they were conducting at Berkeley, with dogs that were being fed on a diet void of calcium. The object primarily was to study pyorrhoea and conditions of the mouth in dogs receiving a diet that was deficient in calcium.

Dr. W. H. Kellogg, Associate Professor of Preventive Medicine, University of California, gave one lecture, the subject being "The Protection of Public Health by Examinations and Immunizations of Milk Handlers."

The balance of the program was furnished principally by members of the faculty of the College of Agriculture, together with veterinarians of California, most of whom were connected with the University of California, the Bureau of Animal Industry, or the State Department of Agriculture.

One evening was given over to an adjourned meeting of the California State Veterinary Medical Association, as is customary. During this meeting routine business was conducted. A special committee of three was appointed to assist the Committee on Legislation, to watch for proposed undesirable legislation that may be introduced during the present session of the State Legislature.

The register showed 152 names. Eleven veterinarians were admitted to membership in the Association and one veterinarian was reinstated, giving the Association a membership of 307.

W. L. CURTIS, *Secretary*.

KANSAS VETERINARY MEDICAL ASSOCIATION

The twenty-fifth annual meeting of the Kansas Veterinary Medical Association was held in Topeka, January 9-10, 1929, with headquarters at the Hotel Jayhawk. There was an excellent attendance, there being approximately 175 veterinarians present out of a membership of 200.

The principal speakers of the first session were Drs. M. P. Ravenel and Homer A. Wilson, of Missouri. These doctors both left their hearers much food for thought.

The second session was opened with a paper on "Infectious Abortion in Cows," by Dr. S. L. Stewart, of Olathe. This subject brought out much discussion, both from research workers and practitioners.

Experimental work on sterility was discussed in full by Drs. E. R. Frank and W. M. McLeod, of Manhattan. This discussion included the work that is being carried on at the Kansas State Agricultural College at Manhattan. The College prepared and presented a motion picture of the technic and results of epidural anesthesia. This was a very well arranged film and demonstrated this form of anesthesia in good shape. Dr. McLeod, of the Anatomy Department, presented the picture, after which Dr. R. R. Dykstra gave a paper on "Local Anesthetics Used in General Practice." Dr. Dykstra went into this subject very thoroughly and gave in detail the action of the various non-narcotic local anesthetics in comparison with cocain hydrochlorid.

The evening session was well attended, nearly 200 being present. Many veterinarians brought their wives and many invited their state senators and representatives who were here for the session of the Legislature.

Dr. R. R. Dykstra was toastmaster. The after-dinner speakers were Mr. O. C. Murphy, of the Kansas City Board of Health; Mr. Wayne Dinsmore, of the Horse Association of America; Hon. J. H. Mercer, State Live Stock Sanitary Commissioner; and Dr. A. T. Kinsley, of Kansas City. Entertainment consisted of piano solos by Prof. Kenyon, of Washburn College, vocal solos, solo dancing and other dancing numbers. After the dinner, a dance was held which was enjoyed by many.

The second day opened with a discussion of "The Difficulties in Raising Little Chicks, Including Feeds and Feeding," by Dr. F. D. Patterson, of the Alabama Polytechnic Institute, Auburn, Ala. This paper was very interesting and well discussed by many present.

"Control of Bacillary White Diarrhea" was the title of a paper read by Dr. E. H. Lenheim, of Burlingame. Dr. J. W. Lumb, extension veterinarian of the Kansas State Agricultural College, gave a paper on "The Workings of the State Accredited and Certified Flocks." "Anaplasmosis in Cattle" was discussed by Drs. P. B. Darlington, A. H. Gish, E. E. Leasure and others.

The final session consisted of a discussion of swine diseases. Dr. Kinsley discussed in full infectious pneumonia in swine, while Dr. C. E. Salsbery discussed and demonstrated, by formalin-hardened specimens, hog cholera, enteritis and flu.

A short business session was held and the following officers were elected: President, Dr. E. F. Kubin, McPherson; vice-president, Dr. L. R. Bruncher, Mulvane; secretary-treasurer, Dr. Chas. W. Bower, Topeka.

Manhattan was unanimously chosen as the place for the next meeting.

Mimeographed copies of the proceedings of this meeting are being prepared and will be mailed to all members in good standing.

CHAS. W. BOWER, *Secretary*.

NEW HAMPSHIRE VETERINARY MEDICAL ASSOCIATION

A joint meeting of the New Hampshire Veterinary Medical Association and the Granite State Dairymen's Association was held at Concord, January 18, 1929. Two papers on the subject of abortion were presented by Dr. W. W. Williams, of Springfield, Mass., and Dr. John M. Buck, Assistant Superintendent, U. S. B. A. I. Experiment Station, Bethesda, Md. The dairymen discussed these papers at the morning session and the veterinarians did the same at the afternoon session. Among the sixteen veterinarians who attended and discussed these papers were Dr. R. W. Smith, state veterinarian of New Hampshire, Dr. F. F. Russell, of Concord, Dr. L. R. Haubrich, of Claremont, Dr. H. R. Clark, of Woodsville, Dr. H. M. Lewis, of Nashua, and Dr. F. S. Gray, of Plymouth.

Dr. E. A. Crossman, B. A. I. Inspector-in-charge of tuberculosis eradication in New England, spoke on the progress being made in the tuberculosis eradication campaign. Dr. R. W. Smith discussed the same question and among other things stated that over 77,000 head of cattle had been tested in New Hampshire during the past year and that of these more than 44,000 head had been tested by local practitioners.

A. L. EDMUNDS, *Secretary*.

LOUISIANA STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Louisiana State Veterinary Medical Association was held at Baton Rouge, January 16-17, 1929. The meetings were held in the Agricultural Auditorium of the Louisiana State University. The meeting was the best attended of any ever held by the Association, in spite of the fact that illness kept a number of the members from the meeting.

The following program was presented:

- "Further Research Work on Anaplasmosis," Dr. G. Dikmans, U. S. B. A. I., Jeanerette.
- "Parasites of Horses and Mules," Dr. Harry Morris, L. S. U., Baton Rouge.
- "Tuberculosis Eradication," Dr. L. B. Ernest, U. S. B. A. I., Washington, D. C.
- "Parasites in Dogs," Dr. E. W. Price, U. S. B. A. I., Washington, D. C.
- "Economic Importance of Poultry in Louisiana and the Veterinarian's Part in Its Development," Prof. C. W. Upp, L. S. U., Baton Rouge.
- "Anatomy of the Fowl," Mr. W. M. Ginn, L. S. U., Baton Rouge.
- "Coccidiosis Complex as Found in Louisiana," Prof. R. L. Mayhew, L. S. U., Baton Rouge.
- "Bacillary White Diarrhea Testing and Its Relation to the Veterinarian," Dr. Harry Morris, L. S. U., Baton Rouge.
- "Methods of Controlling Internal Parasites in Poultry," Clyde Ingram and C. L. Hill, L. S. U., Baton Rouge.
- "Recent Investigation of Feeding Beef Cattle on Grass," Prof. C. I. Bray, L. S. U., Baton Rouge.
- "Points to Be Considered in Selecting a Good Dairy Cow," Prof. C. H. Staples, L. S. U., Baton Rouge.
- "Development of the Swine Department During the Past Year," Prof. J. B. Franconi, Jr., L. S. U., Baton Rouge.

A number of the subjects on the program were presented by the demonstration method.

A banquet was served at the Heidelberg Hotel, the evening of the first day. The program for the afternoon of the second day was put on at the Livestock Pavillon and the veterinarians had an opportunity to look over the live stock, including the poultry, of the institution.

All officers were elected as follows: President, Dr. F. A. Hoell, Mansfield; vice-president, Dr. A. D. Kendrick, Homer; secretary-treasurer, Dr. H. A. Burton, Alexandria.

H. A. BURTON, *Secretary*.

NECROLOGY

JOHN CALVIN CALLANDER

Dr. John C. Callander, of Parkersburg, W. Va., died suddenly, December 9, 1928. Death was due to embolism of the heart.

Born in 1858, in Kirkton, Ontario, Dr. Callander received his veterinary training at the Ontario Veterinary College, from which he was graduated in 1889. After practicing in Canada for several years, he located in Parkersburg and became prominently identified with veterinary work in West Virginia.

Dr. Callander was at one time state veterinarian of West Virginia. He was a charter member of the West Virginia State Veterinary Medical Association and served as president for one term and as secretary for two terms. He was a member of the West Virginia State Board of Veterinary Medical Examiners for one term.

He is survived by his widow, one son and two daughters.

S. E. H.

THEODORE L. BREECK

Dr. Theodore L. Breeck, of Carrollton, Ky., died December 18, 1928, of heart disease. He was 50 years of age. Dr. Breeck was a graduate of the Indiana Veterinary College, class of 1910. He is survived by his widow, his mother, four sisters and two brothers. In addition to conducting a private practice, Dr. Breeck was an automobile sales agent.

D. E. KINSELLA

Dr. D. E. Kinsella, of Peoria, Ill., died at his home, December 23, 1928, following an attack of heart trouble.

Born at Wyoming, Ill., September 25, 1867, Dr. Kinsella attended the Chicago Veterinary College and was graduated in 1894. He was in general practice at Chillicothe, Ill., until about three years ago and in 1914 was appointed assistant state veterinarian of Illinois. For the past three years he had been stationed at the Union Stock Yards, Peoria, in connection with tuberculosis eradication work.

Dr. Kinsella is survived by his widow, two sisters and one brother.

ULYSSES SIDNEY RICHARDS

Dr. Ulysses S. Richards, of Woonsocket, R. I., died at his home, Christmas Day, 1928, a few hours after he had suffered a stroke in his drug store in that City.

Born in Lowell, Mass., March 9, 1874, Dr. Richards attended Lowell common and high schools and then entered the Ontario Veterinary College. He was graduated in 1899 and located in Lowell. During the Boer War, Dr. Richards went to Africa on two different occasions, on government missions. Upon his return he located in Woonsocket and became widely and favorably known throughout Rhode Island and Massachusetts.

Dr. Richards was president of the Rhode Island State Veterinary Medical Association and a member of the Rhode Island State Board of Veterinary Medical Examiners. For fourteen years he served as milk inspector for the city of Woonsocket. He was prominent in the activities of the Woonsocket Lodge of Elks, was a member of the Lions' Club, was prominent in the St. Jean Baptiste d' Amerique organization and was founder of the Young Men's Catholic Association of Lowell.

He is survived by his widow and one daughter.

J. S. B.

CHARLES G. MARXER

Dr. Charles G. Marxer, of Millstadt, Ill., died December 26, 1928. A stroke is reported to have been the cause of death. Born in Millstadt, April 29, 1884, Dr. Marxer attended McKillip Veterinary College and was graduated in 1910. He was in general practice and a member of the firm of Marxer Brothers Mercantile Company. He is survived by his widow, one daughter and one brother, Eugene, formerly mayor of Millstadt.

GEORGE D. GIBSON

Dr. George D. Gibson, one of the most prominent and public spirited citizens of Adrian, Michigan, died early during the morning of December 26, 1928. Death was the result of complications following an attack of pneumonia.

Dr. Gibson was born in Burford, Ontario, Canada, August 29, 1896; the son of Mr. and Mrs. Alexander Gibson. He received his preliminary education in the public schools of Burford, was graduated from the Ontario Veterinary College in 1893 and located at Adrian shortly thereafter.

His genial disposition and enthusiasm for things in which he was interested were outstanding characteristics. He was first president of Adrian's commercial organization; charter member of the Adrian lodge of Elks; a member of the Exchange Club; an enthusiastic and active member of all of the Masonic bodies of his city and a member of the Shrine in Detroit, Michigan. When the Boy Scout movement was revived in Adrian a few years ago, Dr. Gibson became one of its strongest supporters and was later made a member of the Commission and Executive Council of this organization. The establishment of the Scout camp at Washington Lake was largely due to his untiring efforts.

Dr. Gibson joined the A. V. M. A. in 1905 and was a member of the Local Committee on Arrangements for the Detroit meeting in 1916. He was a member of the Michigan State Veterinary Medical Association and he served as president of the latter organization, 1913-1914. He was a member of the Board of Directors at the time of his death.

In 1899, Dr. Gibson and Miss Mamie Wynn, of Adrian, were married, and to them three sons were born. The widow, three sons, one brother and two sisters survive.

B. J. K.

CHARLES E. KINCADE

Dr. Charles E. Kincade, of Charleston, Ill., died at his home, December 28, 1928, after a long illness from various ailments which culminated in pneumonia. He was 58 years of age.

A native of Urbana, Ohio, Dr. Kincade located in Charleston, Ill., a number of years ago and served on the city council for several terms. During the Philippine insurrection he served as an army veterinarian. He is survived by a daughter and two brothers.

ROBERT C. HILL

Dr. Robert C. Hill, of West Alexandria, Ohio, died at the Miami Valley Hospital, Dayton, Ohio, December 27, 1928, following a brief illness from pneumonia and complications. He was 56 years of age.

Following his graduation from the Ontario Veterinary College in 1895, Dr. Hill located in Dayton, Ohio, where he was associated with Dr. Walter Shaw. He later located for himself in West Alexandria, and built up a splendid practice in that community. He had clients in all parts of Montgomery and Preble counties.

Dr. Hill joined the A. V. M. A. in 1911. He was a member of the Ohio State Veterinary Medical Association. He is survived by his widow, one daughter, two sisters and one brother.

WALTER H. PHYFE

Dr. Walter H. Phyfe died at his home in Paterson, N. J., after a brief illness, on New Year's Day. Born in Borina, N. Y., in 1853, Dr. Phyfe attended Union College at Schenectady, N. Y. Following his graduation, he took up teaching and later entered the American Veterinary College in New York City. He was graduated from this institution in 1891 and located at Delhi, N. Y., where he practiced his profession for about twenty years.

Nearly twenty-five years ago, Dr. Phyfe became associated with the Borden's Farm Products Company, as veterinarian, and he served in that capacity continuously until about a year ago, when he retired from active work. Many of the established practices of modern dairy sanitation originated with Dr. Phyfe who was always interested in this work and untiring in his efforts to provide means to improve and safeguard milk supplies.

Dr. Phyfe joined the A. V. M. A. in 1913. He was a member of the New York State Veterinary Medical Society and the Hudson Valley Veterinary Medical Society. His pen on several occasions provided valuable papers for the programs of these meetings.

Dr. Phyfe's kindly personality and sterling qualities endeared him to all with whom he came in contact and he will be greatly missed by his large circle of friends. He is survived by two brothers and two sisters. His wife, Julia Bostwick Phyfe, died about six years ago. A number of his veterinary associates accompanied his remains to his final resting place at New Lebanon, N. Y.

J. McC.

GEORGE H. ROBERTS

Dr. George H. Roberts, of Lafayette, Ind., succumbed to an attack of influenza at the St. Elizabeth Hospital, Lafayette, January 3, 1929. He was Associate Professor of Veterinary Science at Purdue University at the time of his death.

Born in Genesee County, N. Y., November 1, 1864, he attended public schools and Medina Academy.

In 1890, Dr. Roberts left Albion, N. Y., where he had practiced since his graduation from the New York College of Veterinary Surgeons in 1888, and located in Indianapolis. He was one of the founders of the Indiana Veterinary College, which was organized in 1892. For many years he was president of the institution and held the chair of comparative medicine. Dr. Roberts was assistant state veterinarian of Indiana from 1901 until 1913. In 1913, he accepted the position of laboratory director of Pitman-Moore Company and supervised the construction of the biological laboratories at Zionsville. For a number of years, these were devoted exclusively to the production of anti-hog cholera serum. In 1918 Dr. Roberts joined the veterinary staff of Purdue University at Lafayette.

Dr. Roberts joined the A. V. M. A. in 1889. He was fifth vice-president, 1911-12, and first vice-president, 1912-13. In 1912 he was chairman of the Local Committee on Arrangements for the Indianapolis meeting. He was a member of the Committee on JOURNAL, 1915-16, and resident secretary for Indiana, 1917-19.

Himself a student, Dr. Roberts was very successful as a teacher and was highly admired by the hundreds of students who attended his classes at the Indiana Veterinary College during the more than thirty years of its existence. He was equally successful as a general practitioner, attested by the large clientele he had while located in Indianapolis.

Dr. Roberts is survived by his widow, one daughter, one son and one sister. The body was taken to Paxton, Ill., for burial.

F. J. M.

WILLIAM JOHN RATIGAN

Dr. William J. Ratigan, of New Orleans, La., died January 11, 1929, following an attack of influenza. Born at Oswego, N. Y., May 6, 1890, Dr. Ratigan attended New York State Normal School, Ohio State University and the Chicago Veterinary College. He received the degree of D.V.M. from the latter institution in 1914. He served in the Veterinary Corps during the late war and saw service in France. At the time of his death he was conducting a small-animal practice in New Orleans. He joined the A. V. M. A. in 1918.

JAMES H. McMAHON

Dr. James H. McMahon, of Columbia, Tennessee, died at his home, January 12, 1929, following a stroke of paralysis and a long period of failing health.

Born at Spring Hill, Tenn., May 14, 1882, Dr. McMahon attended local schools and then entered the Ontario Veterinary College. He was graduated with honors in 1907 and returned to his home county to practice his profession. Dr. McMahon was a member of the Tennessee State Veterinary Medical Association and enjoyed the unique honor of having held every office in the organization. He is survived by his widow, one daughter, one son and one sister.

W. B. L.

MYRON H. REYNOLDS

Dr. M. H. Reynolds, of St. Paul, Minnesota, died at his home, January 15, 1929. Pneumonia was the immediate cause of death, following an illness which developed the first week in December. He had been professor of veterinary medicine at the University of Minnesota since 1893. He was 63 years of age.

Born in Wheaton, Ill., Dr. Reynolds attended Iowa State College, receiving the degree of B. S. A. in 1886 and the degree of D. V. M. three years later. He then entered the Iowa College of Physicians and Surgeons and was graduated with the degree of M. D. in 1891. The same year he was granted the degree of Ph. D. from the Iowa College of Pharmacy.

After finishing his veterinary course at Iowa State College, Dr. Reynolds spent some time on a large ranch in Louisiana. Later he established himself in general practice at Keosauqua, Iowa. Shortly after this he went to Minnesota, and engaged in Farmers' Institute work under the direction of the late C. C. Gregg, continuing in this work until 1893.

In 1893, he went to the University of Minnesota as veterinarian on the Experiment Station staff and professor of veterinary medicine in the College of Agriculture, located at University Farm. For many years, Dr. Reynolds was a member of the Minnesota State Live Stock Sanitary Board, in the organization of which he played a prominent part. He also served as veterinarian to the Stallion Registration Board of the State of Minnesota for over twenty years. For twelve years (1898-1910) he was a member of the Minnesota State Veterinary Examining Board, and part of this time he served as secretary.

Dr. Reynolds was a prolific writer. He was a collaborator of the American Veterinary Review for many years and was author of "Veterinary Studies for Agricultural Students," a text-book that was used by classes in veterinary medicine in many of the

agricultural colleges throughout the country. He was the author of numerous bulletins and pamphlets published by the University of Minnesota and was a frequent contributor to the programs of veterinary association meetings, both state and national.

Dr. Reynolds joined the A. V. M. A. in 1891 and he was probably the recipient of more committee assignments than any other member of the Association. He was a member (1897-1902 and 1906-1908) and chairman (1908-09) of the Executive Committee. From 1899 to 1904, he was chairman of the Publication Committee and as such edited the annual proceedings of the Association which were published in book form up until 1913. He was a member of the Committee on Intelligence and Education (1904-1905 and 1906-1907); chairman of the Resolutions Committee (1905-1906); member of the Committee of Diseases (1909-1910); chairman of the Veterinary College Investigation Committee (1912-1915); and a member of the Committee on Reorganization of the Association (1915-1916). From 1909 to 1916 Dr. Reynolds served as secretary of the International Commission on the Control of Bovine Tuberculosis. At the Minneapolis meeting, in 1902, he was elected fourth vice-president and on a number of later occasions he was a candidate for the presidency of the Association.

Dr. Reynolds was a charter member of the Minnesota State Veterinary Medical Association and served as president for the year 1899-1900. He was a member of the Minnesota State Dairymen's Association, the United States Live Stock Sanitary Association, the American Medical Association, Alpha Psi and Gamma Sigma Delta fraternities and various Masonic bodies.

Dr. Reynolds is survived by his widow, two daughters and two sons.

W. F. SCOTT

Dr. W. F. Scott, of Los Angeles, Calif., died January 15, 1929, from cancer of the stomach.

Born in Quebec, Canada, in 1860, Dr. Scott was graduated from McGill University in 1885. He practiced in Canada for a few years and in 1890 he went to Chicago, Ill., engaging in practice there until 1917. After leaving Chicago, he went to western Canada and in 1921 moved to California. He is survived by two sons, one of whom is a veterinarian, Dr. R. M. Scott, of Hollywood, Calif.

W. L. C.

Dr. George Fleming McInnes, of Charleston, S. Car., died January 13, 1929. He was the son of Dr. Benjamin McInnes and the brother of Dr. B. Kater McInnes, of Charleston, S. Car., to both of whom our sympathy is extended.

Our sympathy goes out to Dr. F. K. Hansen, of Marquette, Mich., in the death of his father, at Manhattan, Kansas, November 17, 1928; and to Dr. Edward G. Folsom, of Detroit, Mich., in the death of his father, at Mt. Clemens, Mich., January 24, 1929.

BIRTH

To Dr. and Mrs. H. L. Prouse, of Allen, Nebr., a son, Howard Lee, December 25, 1928.

PERSONALS

Dr. R. V. Gibbons (Corn. '25) is now located at 75 Elm St., Clyde N. Y.

Dr. J. H. Lowe (Iowa '00) has been transferred from Helena, Mont., to South St. Paul, Minn.

Dr. E. R. Cushing (Corn. '20) has removed from New Brunswick, N. J., to Plainfield, N. J.

Dr. Wilbur McPherson (O. S. U. '15) has been transferred from Jasper, Fla., to Lake City, Fla.

Dr. A. F. Schalk (O. S. U. '08), of Fargo, N. Dak., was laid up with an attack of flu in December.

Dr. Geo. W. Thurber (Chi. '13), formerly of Necedah, Wis., is now at Stevens Point, Wis.

Dr. J. R. Houchins (U. S. C. V. S. '17) has removed from Bowling Green, Ky., to Franklin, Ky.

Dr. N. F. Ratermann (Cin. '12) is proprietor of the Fort Loramie Hatchery, at Fort Loramie, Ohio.

Dr. R. F. Krenek (K. C. V. C. '14), formerly of Hallettsville, Texas, is now located at Caldwell, Texas.

Dr. C. R. Dilts (O. S. U. '04) has transferred the seat of his operations from Ravenna, Ohio, to Kent, Ohio.

Dr. James E. Patterson (Ont. '25) recently moved into a new home at 14411 St. Marys Ave., Detroit, Mich.

Dr. Russell S. Beardslee (U. P. '28), of Oswego, N. Y., has been appointed Tioga County (N. Y.) Veterinarian.

Dr. F. A. Barker (Ont. '13), formerly of Mt. Victory, Ohio, has located at Belle Center, Ohio, for general practice.

Dr. D. H. Dickie (Mich. '23), formerly of Shelby, Mich., gives a new mailing address: 121 East St. Joseph, Lansing, Mich.

Dr. Daniel W. Gates (U. P. '25), formerly of Howard, Pa., is now located in Bellefonte, Pa. Address: 329 N. Allegheny St.

Dr. B. B. White (K. S. A. C. '20), of San Francisco, Calif., gives us a new address. He is now located at 1870 Jefferson St.

Dr. G. S. Harshfield (O. S. U. '26), formerly at Alliance, Ohio, is now at Canton, Ohio. Address: 1517 Braden Place. N. E.

Dr. George R. Hartmann (K. C. V. C. '11) has been transferred from National Stock Yards, Ill., to Moultrie, Ga., c/o Swift and Co.

Dr. P. T. Engard (Ind. '13), of Marysville, Ohio, was elected a director of the Marysville Kiwanis Club at the recent annual meeting.

Dr. E. F. Sheffield (O. S. U. '22) is assisting Dr. L. J. Proper (Chi. '05), of San Diego, Calif., at the Blue Cross Veterinary Hospital.

Dr. C. C. Harting (Ind. '11), formerly of Crawfordsville, Ind., has opened an office at Waveland, Ind., for the practice of his profession.

Dr. John B. Bryant (Colo. '14), of Mt. Vernon, Iowa, was confined to his home with the flu, for about three weeks, just after Christmas.

Dr. E. M. Dobbs (K. S. A. C. '16), of Berkeley, Calif., has accepted a position as manager of the Alameda County Farm, at San Leandro, Calif.

Dr. Chauncey McCandless (Chi. '15), of Salem, Ohio, has been appointed State Veterinarian of Ohio, by Director of Agriculture, Perry L. Green.

Dr. P. A. Franzmann (Cin. '09) has been transferred from meat inspection at Tacoma, Wash., to Great Falls, Mont., where he is inspector-in-charge.

Dr. T. W. West (Chi. '14) has accepted a position with the San Diego County (Calif.) Veterinarian's office. Address is 739 Fourth St., San Diego, Calif.

Dr. H. J. Shore (Geo. Wash. '11), Laboratory Director of the Fort Dodge Serum Company, was a victim of the flu germ the latter part of December.

Dr. W. S. Plaskett (McGill '93), of Clinton, Mass., is spending the winter in the South. When last heard from, Dr. Plaskett was at St. Petersburg, Fla.

Dr. A. W. Deem (Cin. '18), Logan County (Ohio) Veterinarian, has resigned his position in order to take advanced work in bacteriology at the Ohio State University.

Dr. A. L. Danforth (Corn. '11), of Watertown, N. Y., has given up his practice and is now a special agent of the Bankers Life Company, of Des Moines, Iowa.

Dr. Edward T. Ryan (U. P. '08), of Brookline, Mass., has been commissioned Lieutenant Colonel, Chief Quartermaster 26th Division, Massachusetts National Guard.

Dr. L. O. Leitzman (Ind. '24), who has been engaged in the automobile business for some time, re-entered the practice of veterinary medicine on January 1, at Danville, Ind.

Dr. V. L. Bruns (Colo. '23) has been transferred from Lake City, Fla., to Dade City, same state, on hog cholera control, under the Florida State Live Stock Sanitary Board.

Dr. A. L. Sanders (Ind. '11), formerly of Miami, Fla., and Indianapolis, Ind., has located at Walkerton, Ind., and taken over the practice of the late Dr. Link Grigsby (St. Jos. '18), of that place.

Dr. Kenneth G. McKay (Wash. '21), formerly with the California State Department of Agriculture, has resigned and accepted a position with the University of California, at Berkeley.

Dr. Alvin R. Letson (Gr. Rap. '18), of Fountain, Mich., disappeared from his home on New Year's Day and no trace of him had been found two weeks after his disappearance had been reported.

Dr. Carl L. Martin (O. S. U. '11) has left Southbridge, Mass., to accept the position of Station Veterinarian and Assistant Professor of Veterinary Science at the University of New Hampshire, at Durham.

Dr. A. P. Immenschuh (K. S. A. C. '14) has been appointed County Veterinarian of San Diego County, California. Dr. Immenschuh is Secretary of the San Diego-Imperial Veterinary Medical Association.

Dr. E. D. Martin (O. S. U. '11), of Wilmington, Ohio, who has been conducting tuberculin testing in Paulding County, has been appointed Logan County (Ohio) Veterinarian, succeeding Dr. A. W. Deem, resigned.

Dr. H. L. Simpson (McK. '12), of San Diego, Calif., has returned to his former position with the California State Department of Agriculture, with headquarters at the Los Angeles office, 301 Wholesale Terminal Bldg.

Dr. E. M. Austin (Iowa '18) has accepted a position with the Los Angeles County Live Stock Inspector's office. Dr. Austin had previously been with the organization on a temporary assignment, but this is now a permanent one.

Dr. H. H. Sparhawk (O. S. U. '07), of Detroit, Mich., has been appointed Chief Veterinarian in the Department of Health, Akron, Ohio, and will have charge of the enforcement of the new meat ordinance of that municipality.

Dr. B. H. Branson (Ind. '11), who practiced for eighteen years at Waveland, Ind., and later entered the service of the Federal Bureau of Animal Industry, has resigned his position and returned to general practice, at Rockville, Ind.

Dr. A. D. Paley (McK. '10), of Lake Forest, Ill., recently had a very narrow escape from death when he jumped from his car just before it was crashed by a fast Milwaukee passenger train at the railroad crossing just west of Lake Forest.

Dr. W. A. Johnston (Chi. '10), of Taylorville, Ill., was seriously hurt in an automobile crash about the middle of December. Dr. Johnston suffered a double fracture of the left leg and a number of cuts and bruises on his face and head.

Dr. Ben F. Pigg (Cin. '13), of London, Ky., has been appointed a member of the Kentucky State Board of Veterinary Examiners, succeeding Dr. Edwin Caldemeier (Chi. '11), of Louisville, whose four-year term of office expired July 1.

Dr. J. R. Taylor (Chi. '04), of Sullivan, Ill., has accepted the appointment of Monroe County (Ill.) Veterinarian and entered upon his duties January 1. His contract with the county is for a period of six months, with salary of \$2,000 and all expenses paid. Dr. Taylor will have his headquarters at Waterloo, Ill.